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THE CLINICAL SIGNIFICANCE OF CERTAIN CHANGES IN THE LIMB LEAD ELECTROCARDIOGRAM IN ARTERIAL HYPERTENSION

GILES F. FILLEY, M.D.

From the Medical Clinic, School of Medicine, The Johns Hopkins Hospital

A Clinico-Pathologic Study

This investigation was undertaken to determine what meaning, if any, can be attached to the term "left ventricular strain" as it is used in clinical electrocardiography. During the past 17 years, since Barnes (1) first applied this phrase to the electrocardiographic pattern of inversion of T-waves in Lead I or in Leads I and II, usually with left axis deviation and high voltage, it has become a widely used label for this combination of abnormal changes often seen in cases of arterial hypertension. As such it has proved convenient to the electrocardiographer, but its significance from the clinical standpoint is still a matter of dispute. The present work correlates the clinical and pathological findings in 100 cases of arterial hypertension with these changes in the electrocardiogram.

MATERIAL AND METHODS

The following criteria were considered essential in selecting cases for study and were rigidly adhered to.

1. An established systolic arterial pressure of 140 or over, and an established diastolic level of 90 or over, dating from the time of the first electrocardiogram until death, except for transient reductions during cardiac failure, shock, and terminal moribund states.

2. Admission to the Medical Service[1] of The Johns Hopkins Hospital and a detailed history of previous cardiac symptoms[2] recorded.

3. A complete autopsy on every case.

4. No evidence in the history, electrocardiogram, or subsequent course that any digitalis preparation had been administered within 3 weeks prior to the first electrocardiogram.

[1] One case was admitted to the Urological Service, but was studied by Dr. J. E. Howard.

[2] Any case in which cardiac complaints were indefinite was excluded.

5. A QRS duration of not less than 0.06 nor more than 0.12 seconds.

6. No autopsy evidence of myocardial infarction, and no significant Q-waves in the electrocardiogram.

In the 11,000 autopsies performed from 1925–1945, there were 750 cases indexed as having a history of arterial hypertension. The clinical records of some 500 of these were reviewed and a total of 100 cases was found which satisfied the above criteria. Most of the discarded cases had received digitalis within 3 weeks prior to the electrocardiogram. The selected case histories and autopsy protocols were analyzed in detail with respect to the following data.

1. *Symptoms and Signs of Cardiac Failure*

The duration and severity of these before the first electrocardiogram and the degree of cardiac failure from the date of the tracing until death were estimated. For this analysis the criteria outlined by the New York Heart Association (2) and the physical findings were combined, and 4 categories were established:

No failure: No symptoms and no signs.

Mild failure: Slight dyspnea on exertion only, and/or slight dependent edema, but no rales or orthopnea.

Moderate failure: Dyspnea caused by less than ordinary activity, and/or slight orthopnea and occasional paroxysmal nocturnal dyspnea, and/or moderate dependent edema but no pulmonary rales.

Marked failure: Dyspnea with any exertion, and/or orthopnea, nocturnal dyspnea, marked edema and/or pulmonary rales.

The duration of symptoms and the type of death were combined with the degree of cardiac failure to group the cases for comparative study.

Before First EKG	*After First EKG*
Class A. No failure	Class a. No failure
Class B. Mild or moderate failure 5 months or less	Class b. Mild failure or less than 1 month of moderate; failure secondary or absent at death
Class C. Mild or moderate failure 6–11 months, with no more than 1 month of marked failure	Class c. Moderate or marked failure at least a month, but absent or secondary at time of death
Class D. Moderate or marked failure 12 months or more with at least 1 month of marked failure	Class d. Moderate or marked failure, with death primarily due to cardiac failure

2. *Immediate and Contributing Cause of Death*

The full explanation of the events leading to death was often lacking, especially where several organ-systems were involved. The cases, however, fell naturally into the following groups:

Primary Cardiac Death: Here there were little or no nitrogen retention and no cerebral symptoms. Pulmonary edema was the outstanding feature clinically, and chronic passive congestion at autopsy.

Secondary Cardiac Death: Cases in which there was evidence of cardiac failure, but where uremia or severe infection were important terminal events.

Uremia: Here there was marked nitrogen retention and/or the clinical signs of uremia, but no objective evidence of cardiac failure.

Miscellaneous: Cases in which there was cerebral hemorrhage or thrombosis, infection, cachexia, or exhaustion.

3. *Arterial Pressure at the Time of the First EKG*

In cases where the blood pressure was not recorded on the same day as the electrocardiogram was taken, the average of the pressures a few days preceding and following was used.

4. *Other Clinical Data*

The usual weight was recorded in 97 cases. The age, height,[3] hemoglobin determination, and plasma non-protein nitrogen were recorded in most cases. The serum CO_2 combining power and serum chlorides were recorded in about one-half the cases. The serum calcium was recorded in 15 cases.

5. *Electrocardiographic Definitions*

All electrocardiograms fell into one of the following types, chosen entirely on the basis of the ST-T changes in Leads I and/or II. (See Fig. 1.)

Type I—ST segments showing less than 0.5 mm. depression; upright T-waves.

[3] In 16 cases the length of the body at autopsy was used as the standing height of the patient in life.

Type II—ST segments showing less than 0.5 mm. depression; T-waves less than 1 mm. in amplitude, diphasic, or iso-electric, but not inverted.

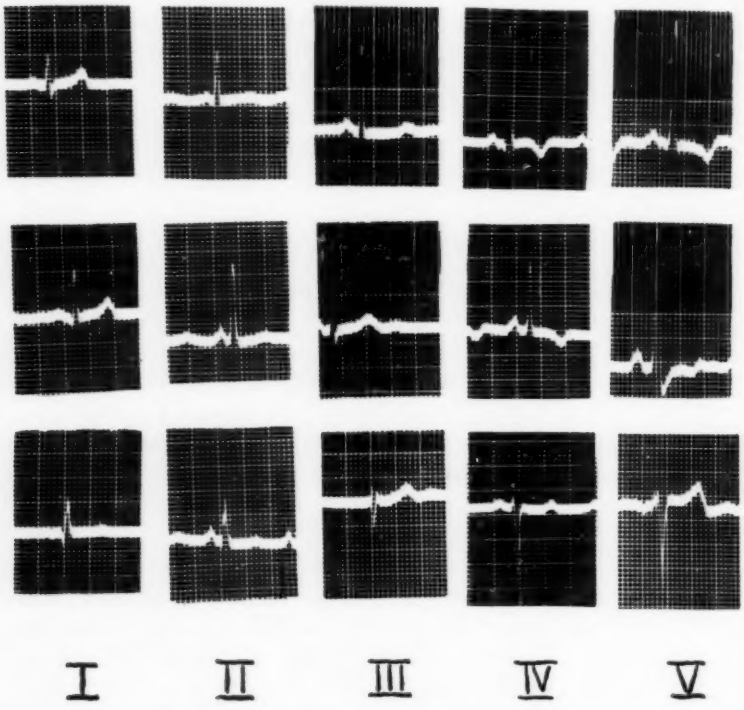

FIG. 1. THE FIVE ELECTROCARDIOGRAPHIC TYPES

Type III—ST segments depressed 0.5 mm. or more; T-waves as in Types I and II.

Type IV—Non-deviated ST segments and inverted T-waves.

Type V—ST segments depressed 0.5 mm. or more and inverted T-waves.

QRS voltage was measured at the sum of R_1, S_1, R_3, and S_3.

6. *Autopsy Findings*

From the weight of the heart in the fresh state and the usual body weight, the percentage increase above normal was calculated according to the data given by Smith (3). The measured thickness of each

TABLE I

Distribution of significant data in 100 cases of hypertension

AGE		ARTERIAL PRESSURE AT TIME OF EKG			EKG		DEGREE OF CARDIAC FAILURE				SURVIVAL	
Group	No. of Cases	Syst.	Diastolic	No. of Cases	Type	No. of Cases	Class Before EKG		Class After EKG		Mos.	No. of Cases
10–19	6		<90	7	I	40	A	30	a	23	<1	43
20–29	11		90–99	13	II	14	B	31	b	26	2–6	15
30–39	24		100–119	26	III	6	C	20	c	24	>6	42
40–49	39		120–139	21	IV	6	D	19	d	27		
50–59	13		140–159	25	V	34						
60–69	7		>159	8								
Avg. 39.5	100	Avg. 192	Avg. 123	100		100		100		100	Avg. 18.3	100

TYPE DEATH	NO. OF CASES	FINAL ANATOMICAL DIAGNOSIS	NO. OF CASES	WEIGHT OF HEART	
				Grams	No. of Cases
Primary cardiac	29	Arterio-and/or arteriolosclerotic nephritis	61	<350	4
Secondary cardiac	19	Generalized arteriosclerosis	16	350–445	26
Uremia	33	Chronic glomerulonephritis	14	450–545	32
Miscellaneous	19	Pyelonephritis	4	550–645	23
		Periarteritis nodosa	3	650–750	12
		Polycystic kidneys	1	>750	3
		Adrenal tumor	1		
	100		100	Avg. 511.0	100

ventricular wall and the pathologist's estimate at gross conference of the degree of hypertrophy of each were tabulated.[4] The degree of

[4] In all cases the thickness of the left ventricular wall was measured at autopsy or described at conference. In 64 cases the thickness of the right ventricle was measured; in 20 cases this measurement was not recorded but there was a con-

coronary plaque formation, sclerosis and narrowing, the presence or absence of hypertrophy of the individual muscle fibers and of myocardial scarring, and the final anatomical diagnosis were analyzed. Table I indicates the case distribution of most of the above data.

RESULTS

The electrocardiographic types were plotted against each of the other items listed above in the attempt to establish correlations, and, where indicated, various non-electrocardiographic data were studied in relation to each other.

A. *Positive Electrocardiographic Correlations*

1. ***Analysis of factors associated with the electrocardiogram:*** Fig. 2 indicates the 6 correlations in which other clinical and pathological data bore a definite relationship to the electrocardiogram. The "strain" pattern was found in about one half of the 77 cases of "vascular disease" showing arteriosclerotic and/or arteriolosclerotic nephritis, or generalized arteriosclerosis at autopsy, and occurred relatively infrequently in cases of glomerulonephritis. The 3 findings which were most frequently associated with Type V were diastolic levels of 120 and over, moderate to marked coronary sclerosis, and markedly increased heart weights at autopsy. It was found that each of these factors was relatively independent of the others so that none could be excluded as a cause of the abnormal ST-T changes.

These 3 factors were studied in the 42 cases with contrasting final deflections whose electrocardiograms were taken within 6 months of death so that changes found at autopsy could be considered probably present at the time of the tracing. The relation of the diastolic pressure to the degree of coronary sclerosis is presented in Fig. 3a. Although there is some tendency for coronary changes to become more marked with the rise in pressure it is not very consistent. This finding confirms the work of Davis and Klainer (4) who found that in hypertension the incidence of coronary atherosclerosis did not increase proportionately as the arterial pressure increased. However, in the

ference statement as to its thickness; in 16 cases it was not specifically mentioned in the notes.

present series a diastolic level of over 120 or moderate to marked coronary sclerosis tended to be associated with abnormal ST-T changes; if both factors were present, a normal pattern occurred in but 2 of

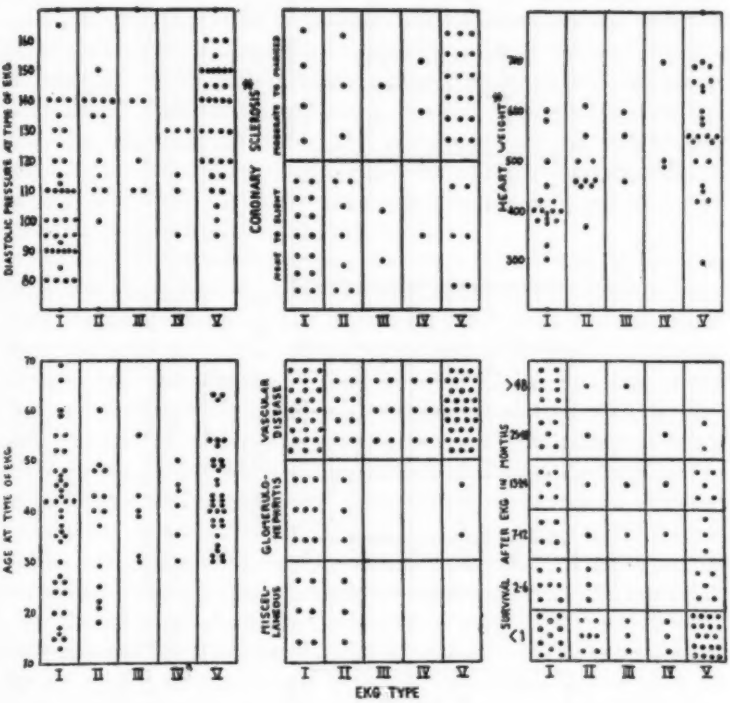

FIG. 2. THE SIX POSITIVE CORRELATIONS BETWEEN ELECTROCARDIOGRAPHIC TYPE AND OTHER DATA

* Only the 58 cases dying within six months of the electrocardiogram are included so that the changes found at autopsy were likely to be present at the time the electrocardiogram was made.

16 cases. On the other hand, all cases whose diastolic pressures were below 120 and who showed insignificant coronary sclerosis at autopsy exhibited Type I electrocardiograms.

In a similar manner the heart weight was not strictly dependent on

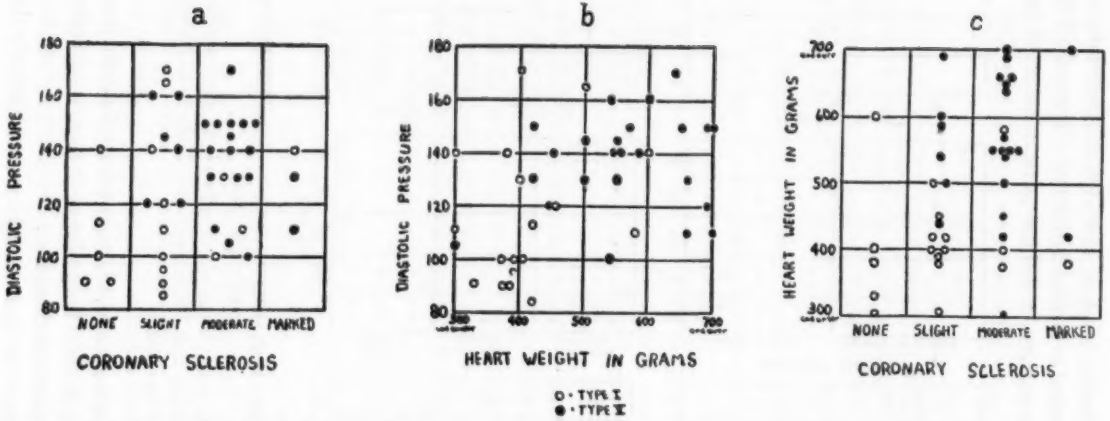

FIG. 3. THE FORTY-TWO CASES SHOWING NORMAL OR "STRAIN" TYPE ELECTROCARDIOGRAMS AND COMING TO AUTOPSY WITHIN SIX MONTHS OF THE DATE OF THE TRACING. CASES WITH TYPE V ARE INDICATED BY SOLID CIRCLES; CASES WITH TYPE I ARE INDICATED BY THE OPEN CIRCLES

the diastolic pressure[5] as indicated by the considerable scatter in Fig. 3b. Again it can be seen that the abnormal electrocardiographic pattern tended to be associated with the coexistence of two factors; in this instance high diastolic pressure and great increase in heart weight.

In Fig. 3c it is evident that there was but little relation between the degree of coronary sclerosis and the increase in heart weight. However, of the 18 cases in which the heart weighed over 400 grams and coronary sclerosis was moderate or marked, 17 showed the "strain" electrocardiographic pattern. The increase in cardiac weight was found to correlate well with the presence of hypertrophy of the individual muscle fibers in the sections of 40 of these cases—a fact in accord with the work of Roberts and Wearn (5).

2. *Analysis of factors influencing survival:* Since it was desirable to determine whether or not the electrocardiogram was *in itself* of prognostic value, it was necessary to exclude other factors which might affect the survival time. Among these age, final diagnosis, arterial pressure and the presence or absence of uremia at the time of the electrocardiogram were found to be the most important. Cases were therefore selected and paired so that they were practically identical in these respects, but which differed in their electrocardiograms, having either Type I or Type V. Four groups of paired cases were analyzed and the results are shown in Table II. It is seen that as the factors which tend to shorten the survival time are eliminated, the abnormal electrocardiograms become less and less ominous prognostic signs.

In the absence of nitrogen retention a very high diastolic level irrespective of the electrocardiogram was found to be the most reliable sign of a poor prognosis. Of 22 non-uremic cases with a diastolic pressure of over 120, 18 were dead within 1 year, with an average survival of 10 months. Of 44 non-uremic cases with diastolic pressures of 120 or below, 22 were dead within 1 year, with an average survival of 28 months.

B. *Negative Electrocardiogram Correlations*

There were 11 clinical and pathological findings studied which bore no relation to the electrocardiographic type, either treated singly or

[5] The lack of correlation between diastolic pressure and heart weight remained unchanged if the latter was expressed as percent above the heart weight considered normal for the given body weight (3).

TABLE II

Four groups of cases individually paired for comparison of contrasting electrocardiograph types

COMPARABLE WITH RESPECT TO:	AGE AND FINAL DIAGNOSIS		AGE AND FINAL DIAGNOSIS AND BP		AGE, FINAL DIAGNOSIS AND ABSENCE OF UREMIA		AGE, FINAL DIAGNOSIS, BP AND ABSENCE OF UREMIA	
EKG Type	I	V	I	V	I	V	I	V
No. of cases	14	14	10	10	8	8	2	2
Average age	41.4	41.2	42.7	42.8	48	48	53.5	54
Final diagnosis	Arterio-and/or arteriolosclerotic nephritis and/or generalized arteriosclerosis							
Average BP	180/108	230/143	214/138	220/136	176/103	208/131	207/117	198/110
Average survival (mos.)	40	5.6	21.5	2.3	42	13	6.5	18
% with moderate or marked cardiac failure before EKG (classes C, D)	21%	50%	20%	40%	13%	25%	0	0
% with moderate or marked cardiac failure after EKG (classes c, d)	50%	50%	60%	30%	63%	63%	50%	50%

TABLE III

Eleven factors studied in relation to ST-T changes and voltage of QRS deflections in which no significant correlations could be established

CLINICAL	PATHOLOGICAL
Duration and severity of cardiac failure before the EKG	Relative thickness of the ventricular walls
Severity of cardiac failure after the EKG	Tortuosity of coronary arteries
Ratio of patient's height to weight	Myocardial scarring
Type of death	Relative degree of sclerosis of large as against small arteries
Plasm NPN	
Serum chlorides, CO_2-combining power and calcium	
Hemoglobin level	

in various combinations. A list of these is given in Table III. Because of their importance 3 of these will be analyzed.

1. *Relation of the electrocardiogram to relative ventricular thickness:* In the cases of this series the routine autopsy measurements and the final statement of relative ventricular hypertrophy were carefully graded. There were 46 cases coming to autopsy within 6 months of the date of the electrocardiogram in which actual measurements of the heart had been made and the degree of hypertrophy of the two ventricles estimated in the conference on the case. In Fig. 4a the ratio of ventricular thickness was plotted against the electrocardio-

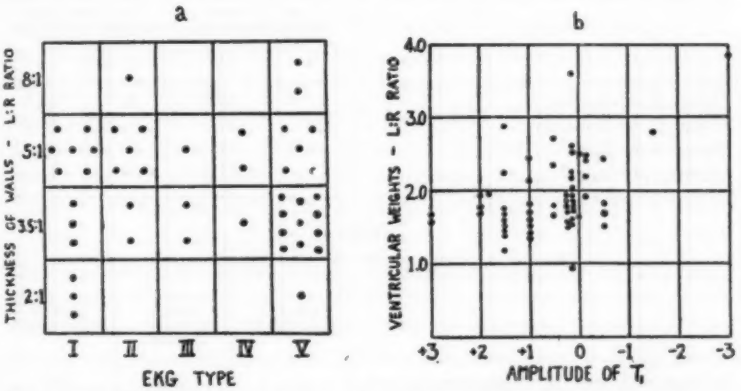

FIG. 4. THE RELATION BETWEEN THE ELECTROCARDIOGRAM AND THE RELATIVE HYPERTROPHY OF THE LEFT AND RIGHT VENTRICLE

graphic type. Of the 13 Type I electrocardiograms, 7 or 54% occurred in association with a L:R thickness ratio of 5:1, while of the 19 electrocardiograms showing "left ventricular strain" only 37% occurred in association with ratios of 5:1 or over.

From the data given by Herrmann and Wilson (6), who correlated the T-wave direction and amplitude in Leads I and II with the relative weights of the two ventricles dissected out at autopsy by a method modified from Lewis (7), a similar correlation study was carried out. In Fig. 4b it is seen that there was poor correlation between relative ventricular weights and the T-wave changes.

2. *The relation of the electrocardiogram to cardiac failure:* In Table

IV the duration and degree of cardiac failure before the electrocardiogram, and the severity of failure after it, do not appear to be related to the electrocardiographic type. Table II indicates that what trends there are tend to disappear if the cases are suitable for comparison. It is noteworthy that the incidence of cardiac failure after the date of the electrocardiogram remains practically identical in the paired cases whether the tracing is of Type I or Type V.

TABLE IV

Correlation between normal ST-T in leads I and II (Type I) and the "strain" pattern (Type V) and the degree of cardiac failure before and after the electrocardiogram

	% OF CASES WITH FAILURE BEFORE EKG		% OF CASES WITH FAILURE AFTER EKG	
	I	V	I	V
Classes D,d—Marked	17.5	26.5	30	26.5
Classes C,c—Moderate	20	20	20	20.5
Classes B,b—Mild	22.5	25	30	32.5
Classes A,a—None	40	17.5	20	20.5

TABLE V

The relation of cardiac failure to 3 other significant data

	AVERAGE DIASTOLIC BP	MODERATE TO MARKED CORONARY SCLEROSIS	AVERAGE HEART WEIGHT
51 Cases with moderate to severe cardiac failure after the EKG (Classes c. and d.)	122	47%	535
49 Cases with none to slight cardiac failure after the EKG (Classes a. and b.)	124	47%	495

3. *The significance of gradations between the normal and the "strain" type of electrocardiogram:* No positive correlations were found between ST depressions in the absence of T-wave inversions, and T-wave inversions in the absence of ST depressions and the other data studied.

C. *The Relation of Cardiac Failure to Other Data*

The 3 factors associated with abnormal ST-T changes, namely high diastolic pressure, coronary sclerosis, and cardiac hypertrophy were studied in relation to the degree of cardiac failure. In Table V it is

seen that there was no tendency for cardiac failure to be associated with very high diastolic pressure nor with coronary sclerosis. It is to be remembered that all cases of myocardial infarction were excluded from this study. The average heart weight was somewhat greater in the cases with cardiac failure.

DISCUSSION

Many of the early papers on this subject were devoted to T-wave changes in general, and inversion of T_1 came to be regarded as an ominous prognostic sign. However, in most studies (8–14, 16) cases of interventricular block, myocardial infarction, etc. were not excluded, a fact which may have contributed to this impression. Barnes (1), in an analysis restricted to T-wave inversions in cases which at autopsy showed "pathological evidence of cardiac lesions" as a basis "for predicating more strain on one ventricle than on the other," clearly demonstrated that the great majority of hypertensive cases selected in this way showed the inversions in Lead I or in Leads I and II, while in mitral stenosis the inversions were characteristically in Leads II and III. Although the investigation was not concerned with clinical evidence of cardiac failure, it was postulated that "when fatigue or strain reaches or exceeds a certain limit, uncompensated metabolic disturbances occur, capable of modifying the electrical forces produced by the right and left portions of the heart and thus to bring about significant inversions of the T-waves." Since then a number of papers (8–17) have appeared which in general indicate that in hypertension the abnormal ST-T changes are associated with a poor prognosis, although Roesler et al (18), carefully excluding digitalis effect, conduction disturbance and myocardial infarction, found little evidence for this conclusion. The term "strain" is often interpreted by the electrocardiographer as indicating myocardial "damage," and in a recent textbook (19) the term "degenerative process" is used. However, Willius (10), in a large group of autopsied cases, found no specific histologic lesions but a high incidence of hypertrophy and coronary sclerosis in cases with inversion of T-waves, and Master (20) concludes that the electrocardiographic changes are produced primarily by the "anatomical configuration of the cardiac chambers . . . and not the condition of the heart muscle." Table VI summarizes pertinent

TABLE VI

AUTHOR	DATE	T_1 AND/OR T_3 INVERTED						T_1 AND/OR T_3 NOT INVERTED (CONTROLS)						EXPLICITLY EXCLUDED			AUTOPSY DIAGNOSIS	FOLLOW-UP METHOD
		No. of hypertensives	Average age	% Deaths	Months survival of those living	% "Cardiac deaths" in total cases	% Sym. &/or signs of cardiac failure before or after EKG	No. of hypertensives	Average age	% Deaths	Months survival of those living	% "Cardiac deaths" in total cases	% Sym. &/or signs of cardiac failure before or after EKG	Digitalis	Myocardial infarction	Interventricular block		
Willius (8)	1922	63	—	74	10.7	72	—	48	—	38	11	27	—	Yes	No	No	No	Letter
Hepburn (9)	1926	36*	54	48	14	—	—	—	—	—	—	—	—	Yes	No	Yes	No	Traced
Willius (10)	1928	73*	56	100	—	59	—	—	—	—	—	—	—	Yes	No	No	Yes	Autopsy
Luten (11)	1928–1929	237†	54	—	—	—	56	—	—	—	—	—	—	No	No	Yes	In 7 Cases	—
Master (12)	1929	55	54	27	36	—	—	57	54	14	36	—	—	No	No	Most Cases	No	"Priv. Record"
Rykert (13)	1935	104*	56	59	17.1	—	90	—	—	—	—	—	—	No	No	Yes	In 20 Cases	"Traced"
van Nieuwenhuizen (14)	1937	62	—	—	—	—	70	91	—	—	—	—	26	No	No	No	In "Some"	—
Rasmussen (15)	1939	24	60	—	—	—	46	31	60	—	—	—	13	No	Clinically	Yes	—	—
Daley (16)	1941	101†	—	50	—	—	—	185*	—	12	—	—	—	No	No	No	No	—
Rasmussen (17)	1945	20	—	70	72	30	—	29	—	21	72	0	—	No	Clinically	Yes	No	Rep. of Other Doctors
Evans (21)	1945	63	40	—	—	—	43	75	40	—	—	—	32	Yes	Most Cases	Yes	—	Post-oper.

* Not all cases were hypertensive.
† T_1 showed changes varying from flattening to inversion.
— Not given.

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studies regarding the prognostic value of the abnormal electrocardiogram, and its relation to cardiac failure. In the studies which have excluded myocardial infarction and interventricular block, and which have included control cases of hypertension with upright T-waves, the abnormal changes tend less and less to indicate early death or cardiac failure.

It is of interest to compare in some detail the present findings with those of Evans (21) and White (22) and their co-workers who studied a large number of hypertensive cases before and after splanchnic resection. Theirs is the only extensive report dealing with an age group similar to that in the present investigation. In their study, as in this one, there were comparable groups with and without abnormal T-wave changes so that comparisons could be made. It was found that there was no significant correlation between the electrocardiographic type and the complaint of dyspnea, a finding confirmed in this report. Evans et al (21) also noted but slight correlation between the size of the heart by teleoroentgenogram and the type of electrocardiogram, a much debated question (15, 18, 23, 24). The suggestion that the lack of definite correlation in the latter instance might be due to the X-ray not being a "sensitive index of enlargement of the heart" is to some extent borne out, since in the present series the abnormal electrocardiograms occurred largely in patients who showed marked increases in cardiac weight at necropsy 1–6 months later, a finding supporting the study of Willius (10), and of Rykert and Hepburn (13).

In view of the correlation between severe coronary sclerosis and Type V electrocardiograms, it is significant in Evans' study that abnormal T-waves were found in a high percent of cases complaining of chest pain. The early view that T-wave changes in hypertension were associated with coronary disease has of course been clarified (13) by the recognition of the differences between the electrocardiogram of coronary occlusion with its ST elevations and the ST depressions of the "strain" pattern, and there has been a tendency to discount the influence of the coronary arteries in producing the T-wave changes in hypertension. The fact that splanchnic resection often results in the disappearance of such changes (22) does not necessarily indicate that structural coronary changes are absent. It is quite possible

* Not all cases were hypertensive.
† T_1 showed changes varying from flattening to inversion.
— Not given.

that the relative sufficiency of these vessels is increased by reducing the work of the heart concomitant with the lowering of arterial pressure.

The problem of prognosis in hypertension is exceedingly complex; and since the advent of treatment by sympathectomy, it has become acute. In the present investigation, evidence is presented that the ST-T changes associated with hypertensive disease do not in themselves carry a grave prognosis, and do not indicate either that the myocardium has failed or is about to fail. There is no doubt that the left ventricle in hypertension is doing more work than in cases with normal arterial pressure, and that in this sense it is under "strain." There is, however, considerable doubt as to the mechanism of left ventricular failure, and, indeed, as to the ultimate cause of death in hypertensive disease, unless it be cerebral vascular phenomena. To what extent the reported findings are at variance with those in the literature (25, 26, 27) depends partly on the method of case selection and partly on the criteria used in evaluating clinical findings. It is believed that the present group of cases represents a fair cross-section of the types of diseases associated with persistent arterial hypertension uncomplicated by myocardial infarction, interventricular block and valvular disease, but including a variety of vascular and renal disorders commonly encountered. It is also felt that a careful study of terminal clinical and pathological findings is necessary before the type of death can be labeled a "cardiac death." In this paper it is estimated that case 52, for example, had mild to moderate cardiac failure for 1 month before the electrocardiogram and none thereafter until the "secondary cardiac death" occurred.

Case 52. H. D., a colored male, aged 40, was admitted with the chief complaint of high blood pressure. The family and past histories were noncontributory. For 1½ years he had suffered from increasingly severe headaches and complained of spots before his eyes. Nine months previously his arterial pressure was 180/134 in the Out-patient Department. There was albuminuria and cylindruria but no signs of cardiac failure. Two months before admission he began to grow weak, to notice dimness of vision, and to vomit occasionally. He had "some dyspnea on exertion" for one month. He had received no digitalis. On examination he was a well-developed somnolent man in no respiratory distress. BP 220/125. There was papilloedema and narrowed retinal arterioles. The lungs were clear. The heart was not enlarged to percussion, an apical systolic murmur was heard. The

second aortic sound was ringing. There was a moderate anemia and leukocytosis. NPN 94; PSP 2%. The heart was not enlarged by x-ray.

On the ward the pulse and respirations were never rapid. Vomiting and amblyopia progressed and he soon became irrational and showed muscle twitchings. The BP remained 175/130 or over until 6 hours before death, but there was oliguria since admission. An electrocardiogram was taken on the fourth hospital day. The lungs were clear to x-ray and physical examination till the day of death when the NPN reached 400. At autopsy there was moderate generalized arterio- and arteriolosclerosis. The left ventricle was hypertrophied but not dilated and there was moderate coronary sclerosis. The lungs were edematous. There was subacute vascular nephritis with necrosis of arterioles.

The electrocardiogram was interpreted as showing "left ventricular strain" in this case.

In case 50 it is estimated that there was mild to moderate cardiac failure for about three years before the electrocardiogram and moderate to marked failure for the four years prior to death, which was primarily due to cardiac failure.

Case 50. M. S., a colored female, aged 40, came to the Out-patient Department complaining of shortness of breath and palpitation on exertion. Her father and one brother had died of heart trouble. The past history was noncontributory. The present illness began with her sixth pregnancy 10 years before when she was told she had high blood pressure. Four months after the birth of her seventh child, the patient was admitted to a hospital because of dyspnea and severe hypertension, and was improved after one month of bed rest and digitalis. She did fairly well for five years taking no digitalis, but progressive dyspnea reappeared and she noticed ankle edema for several months before coming to the Out-patient Department. At that time she appeared well. The lungs were clear. BP 140/90. The heart was enlarged; there were numerous extrasystoles and a marked apical systolic murmur. The liver was not felt, and there was no edema. An electrocardiogram was taken and then she was started on digitalis. For the next four years she suffered from intermittent dyspnea and dependent edema. The BP during this time varied from 180/110 to 220/140. She was unable to do her housework without becoming dyspneic during most of this time. Four months before death she grew much more short of breath and had attacks of paroxysmal nocturnal dyspnea. During her last hospital admission she was constantly orthopneic and dyspneic and showed signs of congestive failure. The NPN was 40 the day before death. At autopsy there was generalized arteriolosclerosis. The left ventricle was hypertrophied and dilated. The lungs and liver showed chronic passive congestion. The kidneys showed little anatomical change.

The first electrocardiogram was normal seven years after the present illness had begun. Later electrocardiograms showed the influence of digitalis. T-waves were never inverted in the limb leads.

In the first case cited above it is felt that despite the burden of marked hypertension for nine months there were no signs of myocardial failure except terminally and that death was primarily due to renal insufficiency. The second case is an example of prolonged hypertension in which cardiac failure was the predominant feature and the primary cause of death.

In evaluating the electrocardiogram in hypertension it is essential to know the stage of the disease at the time of the tracing. In the sixteen cases of this series in which two electrocardiograms not affected by digitalis were taken at least six months apart, there was a definite tendency for the T-waves to become inverted in Lead I and/or Lead II as the disease progressed. It is reasonable to suppose that if the heart fails in hypertensive disease, it will be more likely to fail the longer the duration of hypertension. However, the "strain" pattern evidently reflects primarily the degree of hypertrophy in response to the high diastolic pressure, rather than the state of myocardial sufficiency. Similarly it appears that more prognostic information can be gained from the blood pressure reading and other clinical findings than from the electrocardiogram.

Inversion of T-waves in Lead I or Leads I and II in arterial hypertension has been interpreted by Master (20) as indicating left ventricular preponderance. Katz (28) stated that "ST and T changes are better evidence of great degrees of left ventricular hypertrophy than the axis deviation derived from QRS." Pardee (29) states that the cause of T-wave inversion, whether "ventricular strain or a relative coronary insufficiency . . . is a matter still to be decided." Kaplan and Katz (23) and Langendorf et al (30) have found that when these changes occur in the absence of left axis deviation, the right ventricle is often found to show some hypertrophy at autopsy, but the association is by no means constant. The significance of axis deviation has been under almost constant investigation since the classical work of Lewis (7), and it will not be reviewed here except to point out that the recent use of vector analysis by Ashman and Byer (31) and others indicates that the problem is not a simple one. Since the determination of relative hypertrophy of the left and right ventricles is at best an approximation in routine autopsies, and since the studies in which actual weighing of the ventricles have not correlated ventricular

ratios with ST-T changes in particular, confident dependence on the deflection changes as indices of the degree of left ventricular preponderance appears to be unwarranted. In the present study no significant correlation between ST-T changes and ventricular preponderance could be established.

The demonstration of Barnes (1) that these changes in Lead I or Leads I and II occur frequently in cases of arterial hypertension, and that similar changes are characteristically found in Leads II and III in cases of mitral stenosis is well established. It is possible that the marked difference in the thickness of the ventricles often found in these conditions plays a role in the production of the contrasting electrocardiographic types. That this is by no means established is indicated in a recent study by Wolferth et al (32) who investigated ST segment deviations in experimental animals and who concluded that "in cases of hypertension as with digitalis effect, it seems probable that the source of the disturbance responsible for segment displacement is on the endocardial side." In a given case of hypertension it is evidently not possible to predict from the electrocardiogram the degree of the left ventricular preponderance to be expected at autopsy.

The clinical value of the electrocardiogram in hypertension depends largely on whether or not it can be correlated with cardiac function, past, present, or future. Given an electrocardiogram showing the "strain" pattern, what significant information has been gained? Evans et al (21) showed that it did not indicate that the hypertension was necessarily of long duration and this study shows that the preceding duration and severity of cardiac failure are not reflected. Apparently the degree of left ventricular preponderance can not be inferred. Finally the likelihood of early death or future cardiac failure can not be ascertained from the type of electrocardiographic change alone.

Although the abnormal changes in the coronary arteries at autopsy are but approximations of their functional capacity, the high incidence of coronary sclerosis associated with electrocardiographic changes supports the view that the T-wave inversions in hypertension are associated in some way with the myocardial blood supply. Since the ST-T changes are associated with the heaviest hearts and not with cardiac failure, since it has been shown (5) that in hypertrophied

hearts there are fewer capillaries per unit of muscle, and since sympathectomy often results in a reversion of the electrocardiographic changes to normal (22), the "strain" pattern evidently reflects an anatomical response of the myocardium to the burden of hypertension, rather than a token of its functional weakness. Under the circumstances, the term "strain," as it is used in electrocardiography, need not be interpreted as an unfavorable sign and should perhaps be avoided. It is of interest that White et al (22) conclude that inverted T-waves in the limb leads "appear to be in themselves indications for surgery rather than contraindications."

SUMMARY AND CONCLUSIONS

1. One hundred selected cases of arterial hypertension were analyzed, and the findings correlated with the limb lead electrocardiogram in each case.

2. Inversions of T-waves and ST segment depressions in Lead I or in Leads I and II were associated primarily with high diastolic arterial pressure, coronary sclerosis, and hypertrophy of the heart as a whole.

3. These ST-T changes were not significantly related to an increase of thickness of the left ventricular wall relative to that of the right ventricle.

4. The poor prognosis usually associated with this electrocardiographic pattern was shown to depend largely on other prognostic signs.

5. The pattern of "left ventricular strain" was not in itself an indication that the myocardium had failed or was about to fail.

6. The clinical significance of these findings is discussed.

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524 ABDOMINAL WOUNDS ON THE WESTERN FRONT

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In spite of the steady development of more effective weapons throughout World Wars I and II, it is important for the abdominal surgeon to note that there has been a steady fall in the mortality rate for those abdominal wound casualties who survived long enough to reach operation. A review of the statistics reveals facts that are most interesting to one familiar with the definitive management of such patients at the close of the present European campaign. In view of the most recent results, for instance, it comes as a surprise that, up to the retreat from Dunkirk, a gross mortality rate of about 50% for cases with small bowel wounds was considered satisfactory. Even more startling is the report of Gordon-Taylor (1) stating that during the same period large bowel wounds carried a mortality rate of 60%, which was the same as it had been at the close of World War I. Finally, it is amazing to realize that it was not until 1915 that the expectant treatment of such abdominal wound casualties gave way to surgical exploration.

In seeking to analyze the cause for the improvement in mortality statistics, it is important that many series of cases be recorded in rather minute detail, for it is frequently these small details which mean the difference between success and failure. The omission of such details, which are common knowledge at the time of writing, often handicaps the future reader.

It is to be hoped that the principles of therapy for abdominal wounds, as devised in World War II, will be applied to the treatment of civilian gunshot casualties, for in several series of cases reported immediately prior to 1940, the mortality rates were still in the order of those reported for World War I. (2–8)

MATERIAL

The present paper deals with 524 wounds of the abdomen treated on the Western Front in Europe between 16 September 1944 and 9

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May 1945. Abdominal wounds complicated by injuries to the diaphragm are not included and are being reported in a separate communication. These cases represent those with suitable records treated by thirteen (13) teams of the Fifth Auxiliary Surgical Group which participated in all campaigns of the Ninth United States Army. As outlined in Army directives the teams were charged with the definitive management of casualties admitted to certain installations, principally Field Hospitals.

THE RESUSCITATION OF THE SERIOUSLY WOUNDED

The magnitude of the problem of resuscitation may be seen from Table I, which shows that three (3) out of four (4) casualties exhibited shock in various degrees as evidenced by the systolic blood pressure. The problem was actually more serious than the table indicates, for,

TABLE I

Systolic Blood Pressure on Admission

SYSTOLIC BLOOD PRESSURE	NO. CASES	PER CENT
Over 110	22	27
90–110	21	26
Under 90	37	47

almost without exception, plasma had been given in more forward installations. Intravenous blood and plasma were the mainstays in combating shock. The quantities of these items used per patient steadily increased throughout the war and may have been the most important single fact in the fall in the mortality rate. Ogilvie (9) reported the use of sixteen (16) units of blood and ten (10) of plasma per one hundred (100) casualties at Alamein with an increase to forty-five (45) of blood and twenty-five (25) of plasma per one hundred (100) casualties at Mareth. On the Western Front for similar cases in the present series the comparatively astounding figures of one hundred and ninety (190) units of blood and two hundred and thirty (230) units of plasma per one hundred (100) casualties were reached. These figures do not include the quantities of blood and plasma used subsequent to operation.

A twenty-one-year old casualty of the Eighty-Fourth Division with an abdominal wound complicated by multiple destructive wounds of the extremities received the greatest quantity of such supportive therapy. He was injured at approximately 1700 hours 20 November 1944 and remained at the aid station until 0100 hours 21 November, having received nine (9) units of plasma. He received two (2) additional units of plasma at the collecting company and arrived at the Field Hospital at 0720 hours with unobtainable pulse and blood pressure. There he received two thousand (2,000) cc. of whole blood and by 0945 hours the blood pressure was 110/80 and pulse 80. He was operated upon at 1245, having received five hundred (500) cc. more of blood and four (4) additional units of plasma. At the conclusion of a two-hour anesthetic the blood pressure was 96/50 and pulse 140, the patient having received five hundred (500) cc. of blood and two (2) units of plasma during operation. The total intravenous therapy from the time of wounding until completion of the operation—twenty-one and a fourth (21¼) hours—was 9250 cc. i.e. 17 u. plasma and 3000 cc. whole blood. There was some transient subcutaneous edema which cleared within twenty-four (24) hours after operation and the patient was evacuated to a General Hospital in good condition. This patient illustrated the indispensability of blood transfusion in severe shock from blood loss, for, in spite of eleven (11) units of plasma, the pulse and blood pressure were not obtainable, but after two thousand (2,000) cc. of whole blood his blood pressure was 110/80 with a pulse rate of 80.

The ratio which should exist between the amount of blood and plasma administered became a perplexing problem. Ogilvie (9) recommended two (2) parts of plasma to each part of blood, and the Manual of Therapy for the European Theater of Operations also advised this regime. There is no logical reason for any specific ratio and in the group of cases reported it was finally decided to give the amount of blood necessary to replace the estimated blood loss.

A red blood count and hemoglobin determination were done on the majority of cases soon after admission to the hospital as an aid in establishing the need for blood. This was found to be helpful as many casualties exhibited hemoglobin determination of 60–70% within six (6) hours of wounding. Not a single instance of a red blood count over 5,000,000 or a hemoglobin over 100% was recorded.

Immobilization of the patient came to be regarded as extremely important in preventing shock. Clothes were cut off instead of being removed and even this was not done until the patient had revived. X-rays were not taken until the blood pressure approached normal. Many times it was distressing to observe that turning a patient on his side for a lateral x-ray of the abdomen resulted in a fall of the systolic blood pressure to shock levels again. This problem was solved in one Field Hospital by constructing a narrow wooden frame to elevate the

patient in such a way that the lateral poles of an Army litter would not interfere with a lateral film while the patient was in the prone position. Such a detail as the elimination of unnecessary handling while preparing the patient on the table for operation is sufficiently important to deserve mention.

The jostling, attendant upon ambulance transportation, was undoubtedly responsible for some shock. Individuals without great blood loss, admitted in mild shock (blood pressure 90/60, pulse 100), would often recover if left comfortably alone. The avoidance of this transportation cause of shock is one reason for moving definitive care as far forward as possible and precludes the early post-operative evacuation of the abdominal wounded.

Of lesser importance was the use of oxygen (which was available in sufficient quantity) via B.L.B. masks for severely shocked patients.

Hot water bottles were helpful during the winter months for comfort, but heat per se was not used as a therapeutic measure.

It was not desirable to operate upon individuals whose blood pressure and pulse did not approach normal. However, in some instances recovery did not take place. Unfortunately, records of preoperative deaths are not available, but one can estimate that about three to five per cent of abdominal casualties admitted to Field Hospitals died before operation. Among the records of one of us (H. W. J., Jr.) there are five (5) attempts at prolonged abdominal procedures which were begun with the blood pressure at shock levels. All of these operations resulted in disaster. Although there was much discussion about this point during the campaign, the experience in the present series indicates that successful operations under such conditions must be indeed rare. Abdominal procedures, however, were successfully completed on individuals whose blood pressure fell to shock levels (80 systolic) after the administration of the anesthesia and before beginning the operation.

PREPARATION FOR OPERATION

After recovery from hemorrhage and shock and before operation all cases had the following routine procedures. A levin tube was passed at the earliest practical moment and suction applied before operation and throughout the early post-operative period.

A specimen of urine with reference to blood was required on each patient. Catheterization was used if necessary.

An A-P and a lateral x-ray of the abdomen were considered essential for all suspected abdominal wounds and chest x-rays were also required to guard against surprises in this area.

Morphine was given freely when necessary. In shock morphia should be given by intravenous injection. In a few instances morphia injected subcutaneously was apparently not taken into the circulation due to shock. Under these circumstances a second subcutaneous injection of the drug was ineffectual and dangerous, for with improved circulation both doses were apparently absorbed with resulting respiratory depression. Such patients were particularly difficult to anesthetize as the respiratory rates of six (6) or eight (8) per minute with small tidal exchange often made it necessary for the anesthetist manually to inflate the lungs to force sufficient ether into the patient to obtain relaxation. On more than one occasion it was necessary to delay forty-five (45) to sixty (60) minutes for this procedure.

THE OPERATION

Definitive care for abdominal wounds near the front does not imply operations compromised by incomplete or inferior equipment. With improvisations and additions to the Table of Equipment of Field and Evacuation Hospitals the necessary facilities were developed. The operations were frequently long and tedious. For example, the average operating time in one hundred and fourteen (114) cases was one hour and thirty-six (36) minutes. This does not indicate slowness but rather the extensiveness of the procedure, for, indeed, slow work was not well tolerated, and in no other type of surgery does rapid and accurate operating pay greater dividends.

The importance of good anesthesia cannot be overemphasized. More than 90% of the cases in the present series were anesthetized by nitrous oxide and ether administered through an endotracheal tube with the standard Army portable anesthesis apparatus. Induction by pentothal sodium was common. The remaining 10% of the cases received drop ether or spinal anesthesia. One operator used field block or local infiltration of the wounds with a 1% solution of novo-

caine in conjunction with nitrous oxide and ether to secure greater relaxation and lessen shock.

Morphine and atropin were routinely used before operation.

In this series about 75% of the incisions were rectus or pararectus, 20% were midline, 3% subcostal or transverse, and 2% miscellaneous. Long incisions were almost universally used and various factors determined their placing. It was desirable to place the incision through a portion of the parietes uninjured by the missile to minimize wound infection, and due consideration was given to the possible necessity for a colostomy. As an exact intraabdominal diagnosis was usually impossible, a vertical incision which could be easily enlarged was preferred, however, if the path of the misslie was confined to the upper abdomen, a subcostal type of incision was occasionally used.

Upon opening the abdomen, a large amount of blood was often encountered. If the liver or spleen were uninjured it was necessary to proceed with the routine examination of the abdominal contents to find the bleeding points which could be secured as identified. In our experience, the most common source of bleeding was the small bowel and its mesentery. A standard method of examination of the peritoneal cavity was soon developed. A satisfactory procedure was to begin at Treitz ligament or the ileo-cecal valve and deliver the intestines upon the abdominal wall. Perforations were marked as encountered. A dry gauze held by an Allis Clamp was adequate. Small bowel wounds were then repaired. The stomach, duodenum, liver and spleen were next examined. An investigation of the large bowel was considered best performed with the small bowel eviscerated, proceeding in such a manner that the suspected area of injury was approached last. Finally the bladder and kidneys were examined.

Early in the campaign a mixture of sulfanilamide and penicillin was used in the abdomen and incision. As it was learned that late foreign body reactions were occurring, this procedure was abandoned.

The type of closure varied somewhat, but the most common routine used was a continuous #0 chromic catgut suture in the peritoneum, interrupted catgut sutures in the fascia, and silk in the skin. Stay sutures were required by Army directives. Through and through wire without layer closure was also used with good results.

NEGATIVE LAPAROTOMIES

Of the five hundred and twenty-four (524) cases submitted to operation, four hundred and seventy-nine (479) had injuries to structures of the abdomen. The remaining forty-five (45) cases presented negative findings. In this latter group there was but one death. This occurred in a patient who had been seriously injured by blast and who presented signs so suggestive of abdominal injuries that an exploratory laparotomy was mandatory.

Most of the patients in this category had wounds to the abdominal wall rendering physical examination of the abdomen correspondingly difficult. Wounds of the thorax, fractures of the spine and pelvis, and injuries to the soft parts of the back and flank frequently gave false impressions of abdominal injuries. Since the operation represented so little risk an exploration was always done in doubtful cases.

SMALL INTESTINE

Injury to the small intestine was the most common abdominal lesion encountered. (Table II). Multiple injuries were the rule; a single perforation or laceration occurred in 22% of the cases. The greatest number of wounds sustained by an individual was thirty-two (32), and the average was slightly over five (5). Bleeding from the small bowel and its mesentery was the most common cause of intraperitoneal hemorrhage.

The standard method of treatment was repair. An ileostomy was never used. If a resection was necessary, open end to end anastomosis was the method preferred. The largest number of anastomoses performed in a single case was three (3). This was done in several instances. Many times a single layer of interrupted sutures was used, either silk or catgut, in both repair and anastomosis. This type gave a minimum constriction of the bowel lumen.

Damage to the duodenum occurred in sixteen (16) cases, or 7% of all small bowel lesions, and was associated in every instance with damage to some other abdominal structure. Incision along the lateral attachment and medial reflection of the duodenum provided adequate exposure for the lesions encountered in this small number of cases. The mortality figure of 19% for cases in this group is of the order of magnitude for small bowel injuries in general.

Since no report of a wound to a Meckel's diverticulum was discovered in the recent literature, the following case report is included:

An eighteen-year old German soldier sustained a penetrating wound of the abdomen sometime prior to midnight 28 March 1945. He received two (2) units of plasma prior to admission at 0400 hours 29 March and was in very good condition. He was not operated upon until 1500 hours the same day, having received only one other unit of plasma in the meantime. Operation disclosed multiple perforations of the small bowel, including a two (2) cm. rip in the tip of a large Meckel's diverticulum. The multiple perforations, including the diverticulum, were resected and an end to end anastomosis accomplished. There were also two (2) small perforations of the distal sigmoid. These were treated by closure and proximal colostomy. The patient made an uneventful recovery.

TABLE II

Wounds of Abdomen

STRUCTURE	NO. CASES	INCIDENCE RELATED TO 479 CASES WITH VISCERAL INJURY	PER CENT WITH WOUNDS TO OTHER VISCERA	GROSS MORTALITY
		per cent	*per cent*	*per cent*
Small intestine	218	45	66	17
Large intestine	193	40	74	21
Retro peritoneal hematoma	80	17	48	9
Liver	69	14	71	10
Stomach	51	11	73	22
Negative laparotomy	45	—	—	2
Kidney	42	9	79	29
Evisceration	39	8	79	13
Bladder	28	6	64	14
Rectum	24	5	50	33
Spleen	16	3	69	19
Great vessels	7	1.6	100	86
Gall bladder	7	1.6	100	14
Ureter	3	0.6	100	33
Pancreas	3	0.6	100	33
Common bile duct	1	0.2	100	0

The mortality attendant on injury to the small intestine is given in Table III. Only a very general correlation was found between the number of bowel perforations and mortality rate. Rather, the degree of peritoneal contamination was thought to be the single most important factor in estimating prognosis. Thus, a single transection with spillage throughout the abdomen came to be looked upon as a far more serious situation than numerous holes without leakage.

LARGE INTESTINE

Injuries to the large intestine were only slightly less common than those of the small bowel (Table II).

Exteriorization was the method of treatment. Except for the distal portion of the sigmoid, every area of the large bowel could be satisfactorily exteriorized. That exteriorization, rather than repair, was desirable was adequately demonstrated by the spreading area of gangrene about an exteriorized perforation. Diversion of the fecal stream is not a fundamental principle of simple exteriorization. It is, however, usually desirable to enlarge the bowel wound at the site of exteriorization to prevent obstruction at this point.

TABLE III

Wounds of Small Intestine

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	49	7	14
With wounds to other viscera	96	17	18
With extra abdominal wounds	25	2	8
With wounds to other viscera plus extra abdominal wounds	48	12	25
Summary	218	38	17

If, as in wounds of the distal sigmoid, it is not possible to exteriorize the bowel, a repair may be done followed by a proximal colostomy. Under such circumstances diversion of the fecal stream is desirable and the colostomy may be accomplished by a transection of the bowel at the original operation. This procedure was performed eighteen (18) times.

In a patient whose condition was equivocal, when a relatively large area of bowel had been destroyed or separated from its blood supply, it was sometimes necessary to bring the proximal end of the colon onto the abdomen at a distance from the viable distal end, leaving the reestablishment of the intestinal tract to be accomplished at a later date. This was found to be advisable in four (4) cases of the present series.

In placing the original incision, as has been mentioned previously, it is well to consider the possibility of a colostomy. Although it was

considered preferable to make the colostomy through a separate incision, in not a few instances it was necessary, especially in lesions of the transverse colon, to exteriorize the damaged loop through the operative incision.

The mortality rate for large bowel injuries may be seen in Table IV. It was found that injuries to the cecum were more than twice as

TABLE IV

Wounds of Large Intestine

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	31	4	13
With wounds to other viscera	96	22	23
With extra abdominal wounds	20	3	15
With wounds to other viscera plus extra abdominal wounds	46	11	24
Summary	193	40	21

TABLE V

Wounds of Portions of Large Intestine

PART OF LARGE INTESTINE	NO. CASES	NO. DEATHS	MORTALITY
			per cent
Cecum	27	11	41
Ascending	21	4	19
Hepatic flexure	12	3	25
Transverse	39	6	15
Splenic flexure	21	4	19
Descending	21	5	24
Sigmoid	35	7	20
Not stated	17	0	0
Total	193	40	21

serious as lesions to other parts of the large bowel. (Table V). This may be due to the fact that cecal lesions often result in the leakage of a large amount (1000–2000 c.c.) of liquid intestinal contents into the peritoneal cavity. They are, therefore, comparable to transections of the small bowel with the additional adverse factor of infection. A study of the eleven (11) deaths in the series shows that the majority of these occurred more than twenty-four (24) hours post-operatively in contrast to the more usual experience with abdominal injuries, wherein

one-half of the deaths occur before the first twenty-four (24) hours. The majority of deaths in injuries of the cecum were due to peritonitis.

It is noteworthy that exteriorization of the cecum is physiologically equivalent to a terminal ileostomy. In four (4) instances an ileo-transverse colostomy with exteriorization of the caecum resulted in only one death. On theoretical grounds an operation which discharges the contents of the ileum directly into the transverse colon is to be preferred to one which results in loss of intestinal fluid onto the abdominal wall. Therefore, in simple exteriorization, if the damage to the cecum is so extensive that the fecal stream is lost on to the abdomen, it may be advisable to divert the intestinal contents by an ileo-transverse colostomy.

RETROPERITONEAL HEMATOMA

The third most common finding at celiotomy was a retro-peritoneal hematoma. Perhaps this condition is even more common than Table II indicates, for in cases with major visceral wound repairs it would not be surprising if the occurrence of a small collection of retroperitoneal blood was not recorded in the findings. These bloody extravasations were caused by fractures of the spine and pelvis or wounds of the soft tissues of the back, as well as by direct extension from injured abdominal viscera. That the condition may give signs and symptoms indistinguishable from injury to an abdominal viscus is indicted by the fact that in forty-two (42 instances it was the only lesion found at laparotomy. There is rarely an indication for exploring such a hematoma, the bleeding point can seldom be found, fresh bleeding may be started and often the original bleeding has ceased by the time the abdomen is opened. In all forty-two (42) instances of retro-peritoneal hematoma the incision was closed without drainage. Only one death resulted in this group of cases and that five (5) days after operation in a patient with a compound fracture of the spine, followed by anuria and uremia after receiving a single blood transfusion (Table VI).

LIVER

The third most frequently wounded abdominal viscus was the liver. The surgical problem in liver wounds revolves about arrest of hemorrhage and provision for drainage. The latter is desirable as liver wounds invariably drain bile in quantities related to the size of the

wound. Both objects may be accomplished by the use of a pack. Ordinary Penrose tubing with or without gauze packing, vaseline gauze and the like were used, but in the presence of active bleeding dry gauze was most satisfactory. This was removed as the bile drainage diminished, usually about the tenth day. Light anesthesia was some-

TABLE VI

Retro Peritoneal Hematoma

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	29	0	0
With wounds to other viscera	24	4	17
With extra abdominal wounds	13	1	8
With wounds to other viscera plus extra abdominal wounds	14	2	14
Summary	80	7	9

TABLE VII

Wounds of Liver

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	14	0	0
With wounds to other viscera	36	6	17
With extra abdominal wounds	6	1	17
With wounds to other viscera plus extra abdominal wounds	13	0	0
Summary	69	7	10

times necessary. In this series some of the smaller wounds were neither drained nor packed but the establishment of a drainage tract is regarded as the preferred method of treatment. The drain was brought out through a stab wound, or, if necessary, through the upper angle of the operative incision. In a few instances large blunt needles were used to suture fractured segments of the liver. (Table VII).

STOMACH

Due to the relatively large number of lacerations of the anterior stomach wall, single injuries comprise one-third (35%) of all

stomach lesions. Coincident wounds of the posterior wall occurred in an additional one-third (42%) of all cases.

Repair was the treatment in each instance except one. In the latter case a resection and anastomosis of the Polya type was used. Two layers of sutures were routinely used. Few notes were made as to the approach to the posterior stomach wall, but division of the gastro-colic ligament is a satisfactory way to reach this structure. As with lesions of the small intestine the degree of spillage was the determining factor in making a prognosis. (Table VIII).

TABLE VIII

Wounds of Stomach

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	8	1	13
With wounds to other viscera	23	7	30
With extra abdominal wounds	6	0	0
With wounds to other viscera plus extra abdominal wounds	14	3	22
Summary	51	11	22

KIDNEY

Kidney damage was often minor, as indicated by the fact that out of forty-two (42) cases only six (6) nephrectomies were thought necessary.

Table IX gives the incidence of associated injury. Table X gives the mortality and circumstances under which nephrectomy was done. All nephrectomies were transperitoneal.

Drainage of the perirenal space for relatively minor hematomas was handled differently by various operators. Twenty-two (22) times a drain was inserted through a flank incision. In only one (1) instance is mention made of subsequent drainage of urine. In another case an abscess developed two weeks after operation in spite of the use of drains for eight (8) days. This suggests that it may be unnecessary to institute drainage in the majority of cases.

EVISCERATION

The surgical problem in evisceration is repair of the wound which may be troublesome due to loss of tissue. More than once, at first glance, it seemed that it would be impossible to repair the defect. However, as a matter of experience, it proved feasible in every case to

TABLE IX

Visceral Lesions Associated with Kidney Injury

ORGAN	RIGHT KIDNEY	LEFT KIDNEY	NOT STATED
No. Cases	18	12	4
Liver	12		
Stomach		2	1
Small intestine	5 (2 duodenum)	4	3
Ascending colon	3		
Hepatic flexure	3		
Transverse colon	6	2	1
Splenic flexure		3	
Descending colon		4	
Spleen		1	

TABLE X

Wounds of Kidney

	NO. CASES	NO. DEATHS	MORTALITY	NO. NEPHRECTOMY	NEPHRECTOMY DEATHS
With no other wounds	6	1	17	3	1
With wounds to other viscera	26	6	23	2	2
With extra abdominal wounds	3	2	66	0	0
With wounds to other viscera plus extra abdominal wounds	7	3	43	1	0
Summary	42	12	29	6	3

bring together some tissue to close the gap although at times considerable ingenuity was required. These wounds often exhibited more redness and edema than the surgical incisions, but gave surprisingly little real trouble. Furthermore, peritonitis was not a problem. In some instances bowel perforations were confined to the eviscerated

loops so that intestinal contents were luckily discharged to the outside rather than into the peritoneal cavity. As experience mounted we came to look upon evisceration without anxiety. Tables XI and XII give the details of the organs involved as well as the mortality rate.

TABLE XI

Evisceration

ORGAN EVISCERATED	NO. CASES	DEATHS
Small intestine with perforation	17	2
Small intestine without perforation	2	0
Not stated	9	0
Omentum	6	1
Omentum and small bowel	1	0
Small and large intestine	2	2
Transverse colon	2	0

TABLE XII

Evisceration

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	7	0	0
With wounds to other viscera	18	2	11
With extra abdominal wounds	1	1	100
With wounds to other viscera plus extra abdominal wounds	13	2	15
Summary	39	5	13

BLADDER

The treatment in sixteen (16) bladder wounds was repair with insertion of urethral catheter and supra-pubic tube. Penrose drains in the pre-vesical space were used. In twelve (12) other bladder injuries the wounds were closed and urethral catheters relied upon for drainage. No trouble was encountered in this latter group of cases.

This experience has led us to believe that supra-pubic drainage is not always desirable or necessary in such cases. We have been told by surgeons in hospitals in the rear areas that patients with supra-

pubic drains often arrived in poor condition, especially if there was an associated colostomy. Furthermore, during evacuation, tubes frequently remained in too long, leaving a contracted, infected, encrusted bladder which proved difficult to correct. We, therefore, now believe that if the bladder wounds are small, e.g. bullet holes, and away from the neck of the bladder, it is preferable to close them with a double layer of catgut sutures, and rely on urethral drainage. Penrose tubing into the prevesical space may be used for several days. There were four (4) deaths in cases with bladder injuries, in one (1) of which the bladder was the only visceral wound, but was associated with fractures of the pelvis and femur. (Table XIII).

TABLE XIII

Wounds of Bladder

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	3	0	0
With wounds to other viscera	10	1	10
With extra abdominal wounds	7	1	14
With wounds to other viscera plus extra abdominal wounds	8	2	66
Summary	28	4	14

RECTUM

In this series all wounds of the rectum were extra-peritoneal. They were often associated with destructive lesions of the buttocks and perineum. An exploratory laparotomy, either for rectal wounds or for associated injuries, was considered necessary in each of the twenty-four (24) cases. In all except three (3) a colostomy was done. In seven (7) instances the rectal wound was repaired from below and adequate peri-rectal drainage established. In ten (10) instances adequate drainage was established but no effort made to repair the bowel. In seven (7) instances no drainage beside that established by the missile was thought necessary.

The various methods of treatment may be crystallized into the following principles: (1) adequate drainage from below must be present.

The missile may provide this, but most often it is necessary to enlarge the wound of the entrance. If the hole in the bowel may be easily reached by the finger in the anus, drainage may be established by incising the rectum, including the sphincter, from the injury to the outside. (2) A colostomy should be done in order to minimize widespread perineal infection.

TABLE XIV

Wounds of Rectum

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	5	0	0
With wounds to other viscera	9	4	44
With extra abdominal wounds	7	4	57
With wounds to other visceral plus extra abdominal wounds	3	0	0
Summary	24	8	33

TABLE XV

Wounds of Spleen

	NO. CASES	NO. DEATHS	MORTALITY
			per cent
With no other wounds	3	1	33
With wounds to other viscera	5	1	20
With extra abdominal wounds	2	0	0
With wounds to other viscera plus extra abdominal wounds	6	1	17
Summary	16	3	19

There were eight (8) deaths in the twenty-four (24) cases. This small group yielded the highest mortality rate of any organ, in spite of the lowest incidence of complicating visceral lesions. (Table XIV).

SPLEEN

The incidence of splenic injuries may be seen in Table II. In eleven (11) cases other organs were damaged as follows: stomach four (4), colon eight (8), small bowel three (3), and kidney one (1).

Splenectomy was performed in twelve (12) cases. The four (4) remaining injuries were slight and no treatment was used. The mortality rate is shown in Table XV.

GREAT VESSELS

Injuries to great vessels of the abdomen were rarely seen, but seven (7) cases survived long enough to reach the operating table. In every circumstance there was an associated visceral lesion. The damaged vessels were as follows:

Vena cava below renal	2
Vena cava and both common iliac veins	1
Internal iliac vein, left	1
External iliac vein, left	1
Internal iliac artery and vein, left, and obturator artery, right	1
External iliac artery and vein	1

Ligation was attempted in each instance except in one injury to the vena cava, which was mistaken for the portal vein and suture attempted.

The mortality rate was especially high, but in only two (2) cases did death occur from massive hemorrhage on the table. (Table II).

GALL BLADDER AND COMMON DUCT

The gall bladder was injured seven (7) times, never without liver damage. There were also concomitant injuries to the following organs: stomach two (2), small intestine four (4), including two (2) to the duodenum, large intestine one (1) and pancreas one (1).

In three (3) instances it was possible to insert a tube. Three (3) times a cholecystectomy was done and in one (1) small lesion the perforation was closed. Drains were used in all cases.

In one (1) case there was a complete division of the common duct. This was successfully treated by inserting a small catheter toward the duodenum and drawing together half the circumference of the common duct with interrupted sutures.

URETER

There were three (3) injuries to the ureter, all with damage to other organs. In one (1) instance a cutaneous ureterostomy was done.

This patient died of anuria following a blood transfusion and microscopic examination of the kidney showed hematin in the tubules. In one (1) instance an anastomosis of the ureter was done; however no ureteral catheter was available and the patient was evacuated draining urine from a stab wound in the lower left quadrant. The third wound, near the bladder, was successfully reimplanted into the extra-peritoneal portion of that organ.

PANCREAS

Only three (3) injuries of the pancreas were encountered, all associated with complicating abdominal injuries. The only treatment instituted in these cases was drainage. None exhibited evidence of acute pancreatitis from the injury. There was one death.

POST-OPERATIVE CARE

Under field conditions all but the necessities are eliminated from post-operative care. It was not possible to give special nursing care to unconscious patients; the diet for casualties with multiple sutures of bowel would scarcely conform to civilian practices; and litters cannot be compared with Gatch beds, but the lack of such refinements did not seem to inferfere with prompt recovery. It was necessary to standardize routines and every abdominal case had attached to his field medical record the following mimeographed orders:

1. *Position.* Upon return from the operating room patients will remain flat with head to the side until they are conscious. They may then assume any position but must be turned frequently.

2. *Gastric Suction.* Attach tube to bottle suction apparatus immediately. Be sure that it is working. Check its function every time patient is observed or treated. If it is plugged, irrigate with sodium bicarbonate solution, water or salt solution until it is functioning.

3. *Sedation.* Morphine sulphate, grains one-fourth ($\frac{1}{4}$) subcutaneously every four (4) hours for forty-eight (48) hours unless respirations are below twelve (12).

4. *Penicillin.* Penicillin will be given every four (4) hours, 40,000 units.

5. *Sulfatherapy.* Sulfatherapy begins with the first intravenous

injection. To make sulfadiazine solution, dissolve five (5) gm. ampule of sodium sulfadiazine in 125 cc. of sterile water. This solution contains one (1) gm. per 25 cc. Give 75 cc. (3 gms.) intravenously twice daily.

6. *Fluid Administrations.* Each patient will receive the following basic fluids each day. Additional therapy will be ordered as required.

a. 1000 cc. normal saline solution and 5% dextrose.

b. 2000 cc. 5% dextrose in distilled water.

c. 500 cc. plasma.

These fluids will be given in two (2) treatments at twelve (12)-hour intervals. Fluid intake by mouth is limited to one (1) ounce of water per hour. The first injection of basic fluids begins not sooner than twelve (12) hours after operation.

7. *Laboratory Requirements.* a. Urine output will be measured and twenty-four (24)-hour output will be recorded in the clinical record. b. HBG, WBC and RBC will be measured between forty-eight (48) and seventy-two (72) hours after operation.

8. *Records.* All treatment and fluid measurements, etc., will be recorded on the clinical record at once. All entries will include *Time* and *Date.*

9. *Temperature, Pulse and Respiration.* T.P.R. wull be recorded every eight (8) hours.

10. *Notification of Ward Surgeon.* In event of any change for the worse in the patient's condition, the Ward Surgeon will be notified immediately.

11. *Care of Bladder.* If a patient does not void within twelve (12) hours after operation and at least once each subsequent twelve (12) hours, he will be catherterized.

12. *Dressings.* Dressings will be done by the Surgeon only, except as ordered.

13. *Blood Pressure.* Blood pressure will be taken every thirty (30) minutes for five (5) hours. Notify the Ward Surgeon if the systolic pressure drops fifteen (15) or more points below the highest previous post-operative reading.

MORBIDITY AND MORTALITY

It has been a common practice to correlate mortality with the interval between injury and operation. At the beginning of the war the

six-hour golden period for the operative treatment of casualties was emphasized. Except to control hemorrhage the rational of early operation is to combat infection. In World War II infection has not been a major problem. Penicillin and the sulfonamides have allowed a more leisurely consideration of this factor. In our experience the interval between injury and operation did not greatly influence the mortality. (Table XVI). This means that it is far preferable to delay operation a few hours in order to spend the interim time on the all important problem of resuscitation.

Post-operatively, as before and during operation, the principle danger was from shock. Forty-six (46) of the eighty-three (83) deaths were attributed to this condition. Most of these died within twenty-four (24) hours following operation. (Table XVII). However, not all deaths from shock occurred in the first twenty-four (24) hours, nor

TABLE XVI

TIME ELAPSED BETWEEN WOUNDING AND OPERATION IN HOURS	NO. CASES	DEAD	MORTALITY
			per cent
0–6	15	2	13
6–12	23	4	17
12–24	28	5	18
Over 24	14	2	14

were all deaths in this period due to shock. Of forty (40) shock deaths, six (6) occurred after twenty-four (24) hours. It is in referring to these cases that the term irreversible shock may be used. The six (6) patients exhibited hypotension, tachycardia and usually anuria until death. Four (4) deaths from uremia might also be considered at this time. These patients exhibited tachycardia and anuria for several days, but the hypotension gave way to hypertension in the order of 180/120 until they died in uremia. This latter course of events was seen with other than abdominal injuries and was a particularly dangerous syndrome as, in our experience, there were no recoveries after the period of hypertension began.

The treatment of shock has been considered above and will not be detailed here except to say that after operation hypotension without other signs of shock is not to be regarded as a sufficient indication for

intensive therapy. Such patients will often recover spontaneously, especially if they are voiding normal quantities of urine.

Eleven (11) patients died in uremia following a period of anuria. Four (4) have been considered in the preceding paragraphs. In three (3) the anuria followed the use of preserved blood and autopsy showed hematin deposits in the kidneys. A few other cases had suppression of urine thought to be due to the same cause, but recovered with expectant therapy. Concentrated human albumin was used under these circumstances but the results were difficult to evaluate. Four thousand (4,000) to five thousand (5,000) cc. of intravenous fluid were used

TABLE XVII

Mortality by Cause

CAUSE OF DEATH	NO. CASES	DIED ON TABLE	P.O. DAY OF DEATH—NO. CASES											
			1	2	3	4	5	6	7	8	9	10	11	16
Shock	46	3	37	3	1	1	1							
Uremia	11				1	2	4	1	1	1	1			
Atelectasis and pneumonia	9					1	4	1	1		2			
Peritonitis	8					2	2	1	1			2	1	
Hemorrhage	2	2												
Pulmonary edema	2			1	1									
Blast	2		1			1								
Air embolism	1						1							
Pulmonary embolism	1				1									
Intestinal obstruction	1													1
All causes	83	5	38	4	4	6	12	3	3	1	3	2	1	1

daily unless signs of subcutaneous or pulmonary edema appeared. Two (2) deaths were due to bilateral kidney injuries. One (1) followed a severe crushing injury and one (1) followed a ligation of the vena cava which was thought at operation to be below the renal veins.

Atelectasis and pneumonia were frequent post-operative complications and constituted the principle cause of death in nine (9) cases. It is difficult to give a precise figure for the post-operative incidence of this condition as various observers differ in their requirements for making the diagnosis.

Many surgeons were of the opinion that atelectesis was more common than in civilian life, or, at any rate, bronchoscopy was called upon

more frequently in its treatment. There is no doubt that the classic picture of massive collapse was not uncommon, and many other cases had troublesome bronchial mucus on one or both sides to a degree sufficient to warrant bronchoscopy. This latter procedure was life-saving in some instances. The less effectual back slap was often used.

Casualties were severely ill, and were often not turned frequently, so that it is no wonder that minor elevations of temperature associated with minimal auscultatory findings occurred. It was often difficult to draw the line between atelectasis and pneumonia. As these patients were already receiving penicillin and sulfadiazine, no additional drugs were used, although some form of deep breathing exercise was often advised. In nine (9) deaths in this group all had other complications as wound infection, localized peritonitis and the like.

Pulmonary edema is a pneumonic complication of quite another origin. Two (2) deaths are attributed to this, but other non-fatal cases occurred in this series. One (1) fatal case received nine (9) units of plasma, three (3) units of whole blood, and five (5) units of Alsever's blood (five hundred (500) cc. blood plus five hundred (500) cc. electrolyte), a total of nine thousand (9000) cc. within ten (10) hours. In a sense these cases are indirectly caused by shock which necessitates treatment by intensive intravenous fluid therapy. Expert judgment is required to use adequate fluid on one hand and prevent edema on the other. No rule as to the maximum allowable fluid may be given as tremendous variation in toleration exists from patient to patient.

Peritonitis was the principle cause of death in eight (8) cases. Considering the number of patients with bowel damage, it is a remarkable accomplishment of surgery, penicillin and the sulfa drugs that peritonitis was no more of a problem. Ileus is usually present for three (3) or four (4) days post-operatively. With a small enema or nothing we have learned to expect a spontaneous movement at that time. Only one (1) death was due to mechanical intestinal obstructions, although it might be expected that other cases developed as evacuation proceeded.

An accurate incidence of wound dehiscence is not available from our records, but it is believed to be about one or two percent. No deaths can be attributed to this condition and closure was by through

and through wire stay sutures. Wound infection was not a problem except in rare instances.

The death from air embolism was the result of intravenous air injection following the completion of a transfusion given under pressure. The causes of the other deaths in Table XVI are self-explanatory.

TABLE XVIII

Comparative Mortality Statistics Abdominal Wounds

AUTHOR	CAMPAIGN	NO. CASES	MORTALITY
			per cent
Gordon-Taylor (1)	Pre-Dunkirk 1939–40	276 (small bowel only)	53
		231 (large bowel only)	60
Giblin (11)	Alamein 1942	33	58
		43	28
Ogilvie (9)	Alamein 1942	381	33
Lawdon (12)	North Africa 1942–43	64	44
Estcourt (13)		65	49
Rohlf (14)	North Africa 1942–43	67	12
Surello (15)	Russia	74	32
Nikolaev (16)	Russia	550 (small bowel only)	54
		57 (large bowel only)	74
Jarvis (17)	Italy 1943	130	23
Bastedo (18)	Italy 1943	98	30
Blackburn (19)	Italy 1943	151	34
Jones	Western Europe 1944–45	524	16

In the entire series there were eighty-three (83) deaths, giving a gross mortality rate of 15.8%.

Table XVIII shows the results of similar series. The continuous improvement in a chance for survival cannot be attributed to any one eschelon of therapy. Elliott Cutler (10) summarized the factors in the following list:

1. Improved methods of resuscitation.

2. Better first aid.
3. The use of penicillin and sulfonimides.
4. Improved methods of transport and evacuation.
5. Good general physical condition of the soldier.
6. Standardization of therapy.
7. Better surgery.

It is a fortunate thing that the experience of this war may be applied not only in the treatment of civilian gunshot wounds but, in many circumstances, to the broad field of peace time abdominal surgery.

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EFFECTS OF COCHLEAR LESIONS ON CLICK RESPONSES IN THE AUDITORY CORTEX OF THE CAT[1]

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INTRODUCTION

The resonance theory of hearing assumes that each region of the organ of Corti responds optimally to a particular part of the sound spectrum, and that the pattern of response in the cochlea is transmitted by way of the auditory nerve, central nuclei, and pathways to the auditory cortex.

Evidence for the first part of this assumption, i.e., tonal localization in the cochlea, was obtained by demonstrating that small lesions made in various parts of the organ of Corti cause impairment of cochlear response to specific parts of the sound spectrum (Walzl and Bordley, 1942). Evidence for the second part of the assumption, i.e., spatial projection of the nerve pattern in the cochlea to the auditory cortex, was obtained by demonstrating that electrical stimulation of small bundles of nerve fibers at their points of entrance into the osseous spiral lamina of the cochlea causes responses localized to specific parts of the auditory area (Woolsey and Walzl, 1942).

In an attempt to correlate the results of these two investigations a study was made of the effects of localized lesions of the organ of Corti on responses produced in the auditory area of the cerebral cortex by click stimulation. The results have been published in abstract form (Walzl and Woolsey, 1942), and the completed study is presented below.

METHODS

Cats were deeply anaesthetized with nembutal or phenobarbital sodium, administered intraperitoneally. Both bullae were exposed by separating the structures in the ventral part of the neck, and then the cochlea of one ear was destroyed.

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[1] Aided by a grant from the Ella Sachs Plotz Foundation.

The cerebral cortex contralateral to the intact ear, was exposed. The cat's head was held rigidly in fixed relationship with the stereotaxic apparatus which carried the exploring electrode, and a coordinate system for the brain was established so that observations could be repeated at the same electrode positions.

In each experiment it was necessary to ascertain the extent of the auditory area and to measure the threshold of cochlear response. The auditory area of the cerebral cortex was determined for each cat by means of click stimulation. The cortical potentials resulting from the "click" stimuli were picked up by an exploring wick electrode and an indifferent electrode connected to the scalp. The exploring electrode was moved systematically across the cortex in one or two millimeter steps by means of a stereotaxic apparatus. The location of each point was recorded on a diagram of the cat's cortex which was in scale with the coordinate readings of the stereotaxic apparatus. The potentials, after amplification, were recorded photographically from a cathode ray oscillograph, the sweep of which was synchronized with the click stimulus. An upward deflection of the wave represents positivity in all the charts. Other details of the methods have been described in a previous paper (Woolsey and Walzl, 1942).

A 12 inch speaker, activated by a condenser discharge, was used to produce the "click" stimulus. The speaker used was selected because its frequency range was found to activate the whole auditory area.

Thresholds of cochlear potentials were used to measure the response of the ear. Octave tones from 64 to 8192 cycles were used as stimuli. The thresholds were determined by increasing the intensity of the stimulating tone in steps of five decibels until the amplified cochlear response was just audible in head phones.

A method has been described (Walzl and Bordley, 1942) by which it is possible consistently to obtain impairment of cochlear response to a desired part of the frequency range. This method consists in detaching a small region of the spiral ligament.

The order of procedure followed in most of the experiments was: first, one cochlea was destroyed and a control plotting of the auditory cortex contralateral to the intact ear was made by means of click stimulation Then the thresholds of cochlear potentials for tones from 64 to 8192 cycles were determined for this ear. A small region of the organ of Corti in the apical or basal end of the cochlea was destroyed and the resulting impairment of cochlear response was determined by again measuring the thresholds of cochlear potentials. The effect of the cochlear lesion on the responses of the auditory area of the cortex was ascertained by remapping the area of response to click stimulation. At the end of the experiment, the thresholds of cochlear potentials were again measured to see if there had been any change during the replotting of the auditory area.

In some experiments the thresholds of cortical responses were measured at representative points on the auditory area. This was accomplished by decreasing the loudness of the click to the lowest level at which cortical response could still be elicited.

RESULTS

Cortical Tonal Localization Shown by Cochlear Lesions

In a previous paper, there was shown to be "point-to-point" projection of auditory nerve fibers in specific parts of the cochlea to correspondingly specific parts of the cerebral cortex (Woolsey and Walzl, 1942). The results were interpreted as providing an "anatomical" basis for tonal localization in the higher brain centers. Since then several investigations, of which the present study is one, have demonstrated the reality of frequency localization in the cortex (Licklider and Kryter, 1942; Bailey, von Bonin, Garol and McCulloch, 1943; Tunturi, 1944; 1945).

In the experiments described below, cochlear potentials were used as an index of peripheral auditory function. Therefore, the significance of the results obtained depends upon the relationship of cochlear potentials to the functioning of the auditory system. Considerable evidence indicates that cochlear potentials result from the excitation of the sensory cells of the organ of Corti;and, therefore, their use as an index of impairment of the functioning of the peripheral end organ of hearing is justifiable.

A. Basal turn lesions. The results of an experiment in which a lesion was made in the basal end of the cochlea are summarized in Fig. 1. Five additional experiments with lesions in the basal coil showed essentially similar results.

The control plotting of the cortex (Fig. 1, A) shows that in this animal the auditory area extended, in the anterior-posterior direction, from slightly ahead of the anterior limb of the suprasylvian sulcus to about 1 millimeter caudal to the posterior ectosylvian sulcus; and in the dorso-ventral direction, from the suprasylvian sulcus nearly to the upper end of the pseudosylvian sulcus.

Immediately following the mapping of the auditory area, the thresholds of cochlear potentials were determined for frequencies from 128 to 8192 cycles. The control thresholds are indicated by the zero line in the chart (Fig. 1, C). A lesion was then made in the basal coil of the cochlea by detaching the spiral ligament in the region from 3–6 millimeters from the basal end. This resulted in raising the thresholds for 2048 and 4096 cycles, 25 and 60 decibels respectively, and in abolishing the response to 8192 cycles (solid line, Fig. 1, C).

The broken line in this chart shows the additional change in thresholds during the several hours required for the final plotting of the cortex.

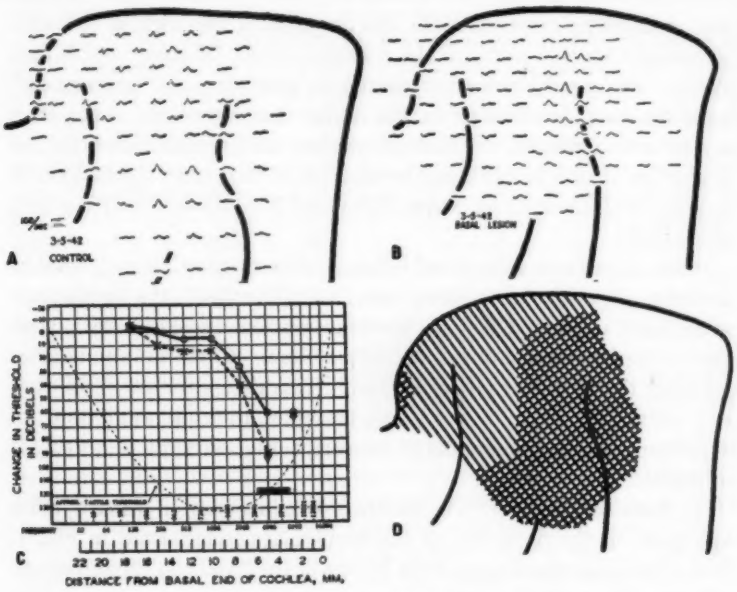

FIG. 1. LESION IN BASAL TURN OF COCHLEA

A. Control mapping of auditory cortex by "click" stimulation. (Fissures shown are the suprasylvian, more or less outlining the area; anterior ectosylvian to left of sylvian; posterior ectosylvian to right of sylvian. Cortical responses are surface positive. Upward deflection of sweep is positive.

B. Repeat mapping after basal turn lesion.

C. Audiogram showing effect of basal turn lesion on threshold of cochlear potentials. (Solid line is impairment immediately after lesion; broken line is additional impairment during period required to remap area. Black bar indicates position and length of lesion in relation to scale at bottom of figure.)

D. Diagram indicating region in which cortical potentials disappeared following lesion.

Fig. 1, B shows the responses occurring in the auditory cortex following the impairment of cochlear response to high tones. This figure and the diagram below it (Fig. 1, D) show that following the lesion there

was disappearance of the positive component of responses in the region above and on either side of the anterior ectosylvian sulcus. The figure also shows that the negative component also persisted. The area in which the positive potentials disappeared corresponds very closely to the area in the cortex where responses occurred on stimulating auditory

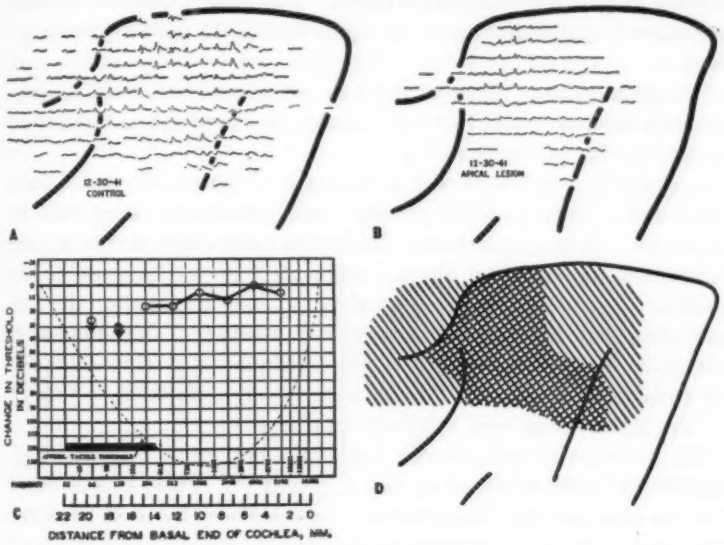

FIG. 2. LESION IN APICAL TURN OF COCHLEA

A. Control mapping of cortex by "click" stimulation.

B. Repeat mapping after apical turn lesion.

C. Audiogram showing effect of apical turn lesion on threshold of cochlear potentials.

D. Diagram indicating region in which cortical potentials disappeared following lesion.

nerve fibers in the basal coil of the cochlea. Conversely, the region in which responses persisted following basal turn lesions corresponds to the response area for stimulation of nerves in the apical turn (Woolsey and Walzl, 1942).

B. Apical turn lesions. The experiment represented by Fig. 2 is typical of the effect of marked impairment of cochlear potentials for low

tones on the responses occurring in the auditory area of the cortex. Four similar experiments confirmed these results.

The control mapping of the auditory area to click stimulation is shown in Fig. 2, A. In this animal potentials were recorded from about 4 millimeters anterior to the suprasylvian sulcus to about 3 millimeters behind the posterior ectosylvian sulcus. The lower border of the area was about 4 millimeters above the upper end of the pseudosylvian fissure.

Threshold measurements of cochlear potentials were made for frequencies from 64 to 8192 cycles. The control readings are indicated by the zero line in the chart, Fig. 2, C.

A relatively long destruction of the organ of Corti in the apical turn was found to be necessary to produce much impairment of response to low tones. In this experiment a lesion was made which started at the helicotrema and extended about 8 millimeters toward the base of the cochlea. Following this lesion no response was obtained for the frequencies 64 and 128 cycles at the maximum intensity of the audiometer (about 30 decibels above the control readings). The thresholds for the frequencies 256 and 512 cycles were raised 15 decibels, but those for the higher tones were relatively unaffected.

The reflection of this low tone loss on the responses of the auditory area of the cortex is shown in Fig. 2, B and D. These figures show that the responses had disappeared or been replaced by small negative potentials in the region anterior to the anterior ectosylvian sulcus and in the upper posterior half of the area as defined by the control mapping. The part of the auditory area in which responses persisted was somewhat C-shaped with a ventral projection extending as far posterior as the posterior ectosylvian sulcus.

C. Duality of cochlear representation in the cerebral cortex. It can be seen from B and D of Fig. 1 that the region in which responses persisted following a high tone loss had an anterior projection in the ventral part of the area; and from B and D of Fig. 2, that the region in which the responses persisted following an apical turn lesion had a posterior projection in the ventral portion. Careful inspection of B, Figs. 1 and 2, shows that the responses in these ventral projections are more or less separated from the rest of the area by regions of minimum response. The position of these ventral projections agrees

with the conclusion reached in a previous paper (Woolsey and Walzl, 1942) that the cochlea is doubly represented in each hemisphere. The dorsal area is designated auditory area I and the ventral one auditory area II. (For comment on terms, see Woolsey and Fairman, 1946.)

If the diagrams D of Figs. 1 and 2 are superimposed, it will be seen that the ventral high tone response area extends well posterior to the ventral low tone response area. This agrees with the conclusion (Woolsey and Walzl, 1942) that the spatial representation of the cochlea in auditory area II is in inverse order to that in auditory area I.

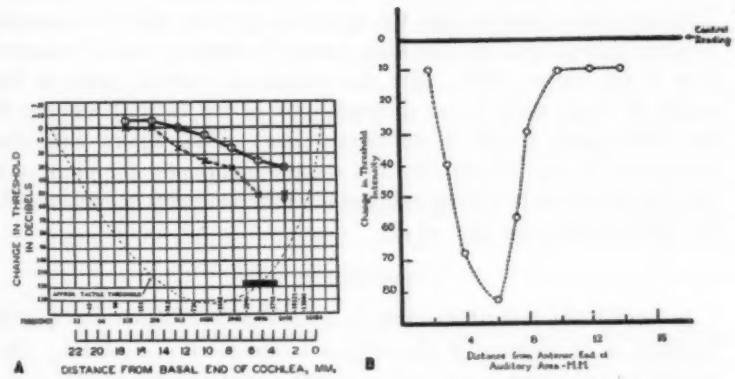
CHANGE IN THRESHOLD IN DECIBELS
22 20 18 16 14 12 10 8 6 4 2 0
DISTANCE FROM BASAL END OF COCHLEA, MM.
A
Control Reading
Change in Threshold Intensity
0 10 20 30 40 50 60 70 80
4 8 12 16
B

FIG. 3. EFFECT OF SMALL BASAL TURN LESION ON THRESHOLD OF CORTICAL RESPONSE

A. Solid line shows effect of lesion on threshold of cochlear response. Broken line shows additional loss obtained by extending the cochlear lesion.

B. Change in threshold of cortical response after first lesion in cochlea.

D. Effect of increasing the degree of impairment of cochlear potentials on cortical responses. In the experiment shown in Fig. 3, the spiral ligament was detached from 4.2 to 6 millimeters from the basal end. This raised the thresholds for 8192, 4096 and 2048 cycles per second by 30, 25, and 15 decibels respectively (solid line, Fig. 3, A). On remapping the auditory area with suprathreshold clicks, no apparent alteration in the amplitudes of the cortical responses or in the extent of the response area was found. However, comparison of the thresholds of response of various parts of the auditory area, taken before

and after making the lesion, showed that the threshold of cortical response in the high tone area in the cortex had been increased (Fig. 3, B). In this figure, the change in threshold intensity of the click stimulus is expressed in volts. On conversion of these values to decibels, the maximum change of cortical thresholds was found to be of the same order of magnitude as the maximum change in thresholds of cochlear potentials (about 30 decibels). Later in the experiment, the amount of high tone loss was increased by extending the size of the lesion (broken line, Fig. 3, A), and then the potentials disappeared from the region which had previously shown only increase in thresholds. This experiment implies that the spread of response which is assumed to occur in the organ of Corti with increase in intensity has its counterpart in the cortex, since, after destruction of a small region of the organ of Corti, there is not disappearance of the cortical response if the stimulating sound is sufficiently loud. This agrees with the experience of deaf people who still recognize correctly the pitch of a tone for which their hearing is impaired if the intensity is loud enough for them to hear the tone at all.

DISCUSSION

As previously indicated, there is considerable evidence for spatial localization of response in the cochlea to the pitch of a sound. The preceding experiments and other recent investigations on the functional organization of the auditory pathways (Ades, Mettler and Culler, 1939; Licklider and Kryter, 1942; Bailey, von Bonin, Garol and McCulloch, 1943; Tunturi, 1944; 1945) demonstrated that the pattern of frequency response in the cochlea is projected to the cerebral cortex. However, in relating these findings to the "place theory of hearing," the effect of changes in intensity of sound on such localization and the question of frequency response in the nerve fibers must be considered.

Several lines of investigation (masking, Fletcher, 1940; summation effect of multiple lesions in the cochlea, Walzl and Bordley, 1942; and response in single nerve fibers to pure tones, Galambos and Davis, 1943) indicate that with increase in intensity, there is spread of the extent of the organ of Corti stimulated by a tone. Several modifications of the Place Theory have been offered to explain pitch discrimination of sounds above threshold. Since the spread is apparently much

greater for low than for high tones, Wever and Bray (1937) have postulated that appreciation of pitch of high tones depends on place, while that for low tones depends on frequency of impulse transmitted by the nerve. Gray (1905) has suggested that pitch discrimination depends on the region of maximum stimulation rather than on a completely local response. Support for this view has recently been obtained by Galambos and Davis (1943) in studying response in single fibers of the auditory nerve. They showed that near threshold each fiber responds to only a narrow band of frequencies, but that as the intensity is increased the fiber responds to additional frequencies. However, they also showed that the tone which excites at threshold continues to act as the most effective stimulus for the fiber (shown by the frequency of impulses in the fiber) up to about 50 decibels. Lorente de Nó (1937) has shown that the external spiral fibers of the auditory nerve send collateral branches to a large number of external hair cells while each radial fiber supplies only a few internal hair cells. Stevens, Davis, and Lurie (1935) concluded on the basis of this demonstration of the simpler innervation of the internal hair cells that these cells are only useful for pitch discrimination of tones near threshold intensity.

It was shown in the preceding experiments that, within limits, destruction of a part of the organ of Corti does not cause disappearance of response from any part of the cortex if the resulting impairment of hearing is less than the intensity of the stimulating sound. One may interpret these results as indicating that with increase in intensity of the sound there is spread of the stimulus along the basilar membrane to uninjured external hair cells which also receive collaterals from nerve fibers which entered the organ of Corti at the level of the lesion. This would result in impulses again being transmitted to the part of the cortex specific for the impaired tone. Since the local lesion completely destroys the internal hair cells, it eliminates these cells from participating in the response to suprathreshold stimulation by the frequency optimal for the end organ at the site of the lesion. No clue has yet been obtained as to the effect of destruction of internal hair cells on hearing. The elucidation of the roles of these cells must await the development of a technique to distinguish between their responses in the cortex and those of the external hair cells.

These experiments indicate that tonal localization in the cerebral

cortex is only as sharp as it is in the end organ, and, as in this structure, the degree of sharpness depends on the intensity of sound. Furthermore, it is probable that the appreciation of pitch at various intensity levels depends on cortical integration of several factors: 1. The place of origin in the cochlea of the nerve fibers stimulated. 2. The type of cells in the organ of Corti the fibers innervate. 3. The number of fibers stimulated. 4. The frequency of response in each fiber.

SUMMARY

Destruction of small regions of the organ of Corti by exposing and depressing the spiral ligament, which previously has been shown to cause impairment of cochlear response to specific tones, was found also to cause disappearance of response to "click" stimuli in specific parts of the auditory area.

Basal cochlear lesions causing loss for high tones were followed by disappearance of response in the anterior superior part of the auditory cortex. Apical lesions causing low tone loss were followed by disappearance of response in the posterior superior and anterior inferior part of the auditory area.

The areas in which response persisted following these lesions confirms the previous conclusion that there are two auditory areas in each cortex and that they have inverse relationships to one another in regard to the various coils of the cochlea.

Cochlear lesions which were too small to cause disappearance of cortical response were found, however, to increase the threshold of cortical response in appropriate parts of the auditory area.

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BOOK REVIEWS

Agnosia, Apraxia and Aphasia. By J. M. NIELSEN. 2nd Ed. Illus. 292 pp. $5.00. *Paul B. Hoeber, Incorporated, New York, New York,* 1946.

This book presents the results of a recent study of aphasia and related disorders from the point of view of cerebral localization. The authors' approach is similar to that of Henschen and differs sharply from that of most recent writers who have taken a more psychological attitude. The voluminous literature is carefully analyzed and the author's own large experience is given. It is astonishing how much has been compressed in these few pages. The presentation is lucid. The most novel idea presented is the emphasis upon the function of the minor cerebral hemisphere in recovery from aphasia. Unquestionably Nielsen has clarified the subject and has made an important contribution to the study of these complex disorders. It is one of the most interesting books the reviewer has had the pleasure of reading for a long time. Every neurologist, neurosurgeon and psychiatrist should have one.

F. R. F.

The Bacterial Cell. By RENÉ J. DUBOS. 460 pp. *Harvard University Press, Cambridge, Massachusetts,* 1945.

Before the reader has finished the first chapter of this book he becomes more more than ever aware of the extreme complexity of this microscopic form of life, and long before the end he marvels at the way in which these complexities have been integrated into a unified picture of what constitutes a living bacterial cell. This is far from a textbook of bacteriology, in which the various types of organisms are described specifically and in turn. Rather, it is one in which the many phases of bacterial life are dealt with in general terms and specific examples used liberally to illustrate the points involved. The author has managed remarkably well throughout to keep the spotlight focused on his primary objects, while the consequences of the growth of these organisms in the body, with the resulting infections and immune reactions are only considered in passing and as they help us to understand the properties of the specific bacteria themselves.

In the early part of the book, in which most of the discussion concerns the cytology of bacteria as well as their physicochemical and staining properties, the structural details of the organisms are correlated as nearly as possible with their multiple functions. The tubercle bacillus may be taken as an example. It is usually spoken of as having a waxy capsule which accounts for many of its peculiarities, but the author finds evidence to show that the known high lipid content of this species is not present merely on the surface, but instead is evidently intimately bound to the cellular structure itself. On the other hand the capsular material which can be demonstrated on virulent strains of many bacteria—notably pneumococci, Friedlander bacilli and influenza bacilli—is almost entirely on the outside and probably consists of material that has been excreted by the cell rather than an

integral part of the cell itself. The manner in which enzymes may be employed to study the cytology of bacteria is demonstrated by the destruction of the capsular material of pneumococci and streptococci by specific enzymes and the inactivation of the M-substance of Group A hemolytic streptococci by trypsin. Such reactions take place without killing the bacteria and suggest that these substances are all located superficially. Also, the destruction of the Gram-positive properties of bacteria by ribonuclease makes it seem possible that ribonucleic acid on the surface of the bacteria may be responsible for the Gram-positive reaction.

In a consideration of the nature of virulence of bacteria, Dubos reminds us that this property has to be regarded in the light of several qualifications and under the varying conditions that may lead to infection. The variables involved are the strain of the bacterial type, its growth phase, the host in which the infection takes place and the conditions under which the organism is introduced into that host. What he stresses in this regard is the significance of the host-parasite relationship rather than that of the micro-organism per se. Some characteristics of bacteria which are of importance in determining whether or not they can establish an infection are their resistance to the normal and immune mechanisms of the host, their invasiveness, the production of toxins, etc. In respect to artificial immunization, the author points out the relative importance of certain fractions of the bacterial cell, in contrast to that of the whole intact organism. The evidence suggests that the surface components, such as the pneumococcal capsular polysaccharides, the M-protein of Group A streptococci and the O and Vi antigens of the Gram negative bacilli, are the most significant fractions from that standpoint.

In the chapters on bacteriostatic and bactericidal agents, the author analyzes the several factors which render bacteria more susceptible or more resistant to antiseptics and antibiotics. Thus, the high phospholipid content of certain types—notably the Gram negative organisms—probably plays an important part in their protection against certain toxic agents. Experimental evidence for this is found in the fact that minute amounts of cephalin can completely protect Gram positive bacteria from the lethal effect of gramicidin. The acid or basic properties of the cells are also of importance in this regard; the Gram positive species being more susceptible to the basic dyes than the Gram negative ones. This may be due to the fact that their cellular material is very acidic, while the opposite is true of the Gram negative bacteria. The age of the culture is another factor, for it is well known that young cells are more susceptible to all sorts of toxic agents than are the older forms; this has certainly been found to be true in their reaction to penicillin. Likewise, some bacteria can overcome the inhibition of growth due to substances which interfere with certain metabolic functions, by employing a different type of metabolic reaction; and others are capable of producing variant forms which may be more resistant than the parent cell.

There is a discussion of the types of inhibition caused by substances capable of competing with metabolites which are essential to bacterial growth, and the sulfonamides may be examples of such antibacterial agents. Not much is said specifically about the mechanism of penicillin's anteribacterial action, but the author

evidently agrees with Hotchkiss, who pointed out the fact that whereas most agents toxic to bacteria appear to affect the process by which these cells liberate energy, the sulfonamides and penicillin probably chiefly affect the way in which the susceptible cells *use* the energy obtained from the substrate on which they are growing. An important cause of drug fastness is thought to be the formation of variant forms, and the possibility is mentioned that the wide-spread use of chemotherapeutic agents may favor the production of many variants. These new forms may be dangerous from the standpoint of pathogenicity and resistance to chemotherapy. This is certainly an apt observation at a time when these drugs are being used in such a widespread and uncritical manner.

Finally the author expresses the hope that more specific chemotherapeutic agents may be found which would be capable of attacking the organisms directly rather than by affecting their metabolic functions. Purified forms of enzymes, if and when prepared, might answer this purpose by specifically destroying the structural components of bacteria that are of particular importance in producing infections.

There is an addendum by C. F. Robinow of London with some excellent photographs concerned with the "nuclear apparatus and cell structure of rod-shaped bacteria."

An index is also included as well as a bibliography of seventy pages which this reviewer feels must cover the situation quite thoroughly. The author is to be congratulated on the clarity of his writing and on the critical manner in which he has dealt with the many controversial views in this field. The book will undoubtedly take its place as one of the classical contributions to the science of bacteriology.

A. M. F.

A Bibliography of Infantile Paralysis with Selected Abstracts and Annotations. By LUDVIG HEKTOEN and ELLA M. SALMONSEN. 672 pp. $15.00. *J. B. Lippincott Company, Philadelphia, Pennsylvania,* 1946.

Numerous devices have been developed in recent times to alleviate the almost hopeless bibliographic plight of the modern research worker. In this volume, the medical investigator and practitioner now have available an exhaustive bibliography of a single disease, compiled in a rather novel fashion. The entire literature of the disease, since its description by Underwood in 1789, is arranged chronologically with selected abstracts and annotations liberally supplied, especially in the more recent literature. In addition to the chronological arrangement, a comprehensive index makes the material available for subject and author reference. 8320 references up to 1944 are included and supplements are promised for later years. It is interesting to note that of 8320 references, 6988 were published in the years following 1908 when Landsteiner published the first report on successful transmission of the disease to monkeys. Half of the references date from 1926. Abstracts are generously supplied for references in the period following 1932, the year of publication of the comprehensive review of the International Committee for the

Study of Infantile Paralysis. Abstracts examined by this reviewer were of excellent quality. Workers in this field must be grateful to the compilers and editors of this volume, and to its sponsors, for an important contribution to the poliomyelitis literature. The book is beautifully printed, and the paper and binding are excellent.

D. B.

Diagnosis and Management of the Thoracic Patient. Edited by CHARLES P. BAILEY. Illus. 334 pp. $4.00. *J. B. Lippincott Company, Philadelphia, Pennsylvania*, 1945.

This book contains a series of 14 articles on thoracic surgical subjects by various well known specialists. Many of these chapters represent the best on the particular subjects considered. The character of the contributors is gauged by the names of Aycock, Ochsner, Head, Sweet and others. The subjects considered include 5 chapters on surgical tuberculosis, one on primary bronchogenic carcinomia, three on chest infections (bronchiectasis, lung abscess, and emphyema) and 5 on other thoracic subdivisions. The charts are good but the roentgenograms do not reproduce satisfactorily on the thin paper used.

This series of essays is one of the most up-to-date on the subject of thoracic surgery.

H. N. H.

Diseases of the Skin. By GEORGE CLINTON ANDREWS, A.B., M.D. 3rd Ed. Illus. 937 pp. $10.00. *W. B. Saunders Company, Philadelphia, Pennsylvania*, 1946.

This well turned out book, consisting of 937 clearly printed and well illustrated pages, may be classed among the medium sized dermatological texts.

Too small to be entirely comprehensive, still it is too large to be used as a hand book by the student at work in the clinic. It should be justifiably popular among those who are appalled by the greater size and cost of the more exhaustive tomes.

The subject matter in this edition has been revised, to some extent rewritten, and enlarged to contain diseases and therapeutics missing in the older printings. Discussions, however, are still of a very general nature, leaving in many instances, much to be desired by the critical reader.

There is a lack of emphasis upon certain common diseases, such as, "Housewife Eczema," for example, and a tendency to confuse by a critical approach to the usages of the terms eczema and dermatitis, which I believe have been more rationally classified elsewhere.

Then, too, in a work of such admittedly great general worth, it is regrettable to see perpetuated ancient nostra such as the dietary treatment of acne vulgaris, including spurious etiological factors such as intestinal putrefaction and fermentation.

Sulfonamides are frequently recommended for topical applications, without sufficient emphasis upon their inherent dangers on the one hand; and the specificity of sulfapyridine in the treatment of Dermatitis Herpetiformis is not stressed, on the other.

Diseases of the vulva are not clearly delineated. This is noticeable particularly in the accounts of herpes, monilial vaginitis, and the differential discussions of lichen planus and kraurosis vulvae.

A fairly complete bibliography is present, and the illustrations both clinical and histopathological are excellent.

H. H. H.

Evolution of Plastic Surgery. By MAXWELL MALTZ. 368 pp. $5.00. *Froben Press, New York, New York,* 1946.

In attempting to review the evolution of plastic surgery the author is confronted with the fact that a large portion of the surgery performed up until the advent of anesthesia and asepsis may be classified as plastic surgery and that such a review in reality tends to include the development of the whole field of surgery. Most of the material in this section of the book, however, holds fairly well to the theme of plastic surgery, or at least to rhinoplasty, and presents many interesting historical facts. Certain sections are poorly organized or poorly titled. The chapter on free skin grafts, for instance, contains material on the technique of rhinoplasty, repair of cleft palate, flap grafts, and plaster of Paris dressings.

In surveying the more recent work in plastic surgery the truly fundamental and important advances are not adequately emphasized and are at times less thoroughly discussed than are minor technical improvements. The lengthy discussions of some of the author's relatively inconsequential techniques seem out of proportion. Included are preoperative and postoperative photographs illustrating some of the excellent results the author has obtained in the treatment of certain common deformities.

Some of the information is misleading; for instance it is stated (page 293) that the utilization of small deep grafts has been found highly effective in treatment of war wounds and burns particularly of the face and hands.

It is thought that the chief value of this book lies in the compilation of interesting historical facts relating to plastic surgery, particularly those in ancient times.

W. L.

Fractures, Dislocations and Sprains. By J. A. KEY and H. E. CONWELL. 4th Ed. 1322 pp. $12.50. *C. V. Mosby Company, St. Louis, Missouri,* 1946.

With its first edition in 1934 "Key and Conwell" became the most universally accepted American text on traumatic orthopedics. The fourth edition, larger than its predecessors, will maintain its preeminent position. The section on fractures of the hip has been brought up to date with illustrations and discussions of the newer operative methods for femoral neck and intertrochanteric fractures. A section on war surgery has been added. It has on the whole a rather pedantic tone, remote from the battlefield. Secondary wound closure is briefly discussed, vaseline gauze is still recommended, and little is said of the fundamental change in surgical treatment of the Orr-Trueta principles of after-care in favor of active intervention.

M. M. R.

Hospital Care of the Surgical Patient. By GEORGE CRILE, JR., and FRANKLIN L. SHIVELY. 2nd Ed. 288 pp. $3.50. *Charles C Thomas, Springfield, Illinois,* 1946.

This excellent handbook for the young surgical house officer and senior medical student is particularly successful in its common sense treatment of the problems of postoperative care. Discussion is pointed, simple, and practical. There is a sound preliminary discussion of physiologic principles. However, discussion of cardio-respiratory physiology is omitted. The discussion of shock unnecessarily denies the serious plasma loss from large wound surfaces and resultant shock.

One has the feeling that the book is harmed by an occasional descent to too elementary a level. The descriptions of procedures are detailed and the procedures themselves too elaborate.

The section on blood transfusion is obsolete—the only method described is the open vessel citrate method described by Lewisohn in 1915. Issue might also be taken with the statement that there is no treatment for the urinary suppression of hemolytic transfusion reactions.

There is a new section on the treatment of wounds.

M. M. R.

Motor Disorders in Nervous Diseases. By ERNEST HERZ and TRACY J. PUTNAM. 184 pp. $3.00. *King's Crown Press, New York, New York,* 1946.

The authors' original plan was to prepare a collection of films for teaching neurology. They found before long that it would be helpful to provide a descriptive syllabus to study before and after viewing the films. This little booklet is the result. It consists of a brief discussion of the common motor disorders with a few anatomical diagrams and many reproductions of short strips of the films. The booklet costs $3.00; the set of 10 films, $450.00.

F. R. F.

Principles and Practice of Tropical Medicine. By L. EVERARD NAPIER. 917 pp. $11.00. *The Macmillan Company, New York, New York,* 1946.

This book has particular interest for internists in the United States for two principal reasons. Soldiers returning from overseas have brought home with them many kinds of tropical disease and, which is equally important, large numbers of medical officers while in the United States Army and Navy have had a taste of studying and treating tropical diseases in many parts of the tropical world. The author, Dr. Napier, after years of firsthand experience in the Calcutta School of Tropical Medicine, has served as visiting lecturer or visiting professor in four large medical schools in this country. He needs no introduction to students of tropical medicine.

Modern teaching of clinical medicine depends to a large degree on clinical material in the form of patients suffering from various diseases. In recent years improved sanitation and public health has gone far toward eradicating most of the

diseases which are commonly found in the crowded and relatively uncivilized tropical areas of the world. This has resulted in wide-spread ignorance on the part of United States physicians concerning even such a common disease as malaria, while Kala-Azar and Leishmaniasis and various fluke infections are apt to leave them gasping. Dr. Napier's book brings these diseases before the reader in a factual and highly systematic fashion. Particularly well done are the chapters on malaria, Kala-Azar, yaws and schistosomiasis.

It is unfortunate that Dr. Napier has been unable to include in this edition any of the vast amount of research and clinical observation that has been reported in the past few years. Doubtless he already is preparing to bring up to date particularly those sections dealing with the treatment of malaria and the chapter on Tsutsugamushi fever.

This is a good book. The discussion of personal hygiene in the tropics, for instance, is simple and straightforward but authoritative. The descriptions of diseases and their treatment is presented in a charming, discursive style which makes it very easy to take. This book should be available to doctors and students who encounter problems of tropical medicine.

H. M. T., Jr.

Roentgen Diagnosis of Disease of the Gastro-intestinal Tract. JOHN T. FARRELL, JR. Illus. 271 pp. $5.50. *Charles C Thomas, Springfield, Illinois,* 1946.

This well organized presentation of the diagnosis of diseases of the digestive tract is arranged on the basis of the Standard Classified Nomenclature. Debatable subjects are purposely avoided in the attempt to make a straight-forward presentation of the subject as the author sees the basic facts. He recommends further reading for amplification. This is a natural outcome of the development of the book from a teaching manual which has been expanded and amplified.

The only notably under-emphasized point is the extreme difficulty which all fluoroscopists are experiencing in determining the state of affairs or presence or absence of pathology in the gastro-intestinal tract which has been subjected to surgery. These cases become more numerous all the time and present very confused pictures. Resections have been made at higher and higher levels and total gastrectomy is done more and more frequently. In both types it is usually impossible to palpate the areas most concerned and it may even be impossible to apply indirect pressure. Thus all conclusions must be drawn from fluoroscopy without palpation and from films.

W. H. B.

Surgical Treatment of the Motor Skeletal System. Edited by FREDERIC W. BANCROFT and CLAY RAY MURRAY. Illus. 2 Vol. $20.00. *J. B. Lippincott Company, Philadelphia, Pennsylvania,* 1945.

This work is published in two volumes. The first deals with Congenital Malformations and Anomalies; Acquired and Static Deformities, such as Genu Valgum,

Pes Valgoplanus and Scoliosis; Paralytic Disorders; Affections of Back, Muscles, Fasciae, Tendons, Bursae and Ganglia; New Growths; Diseases of Bones and Joints and Amputations. The second volume considers Fractures and Dislocations in General; Fractures and Dislocations of the Trunk; Fractures and Dislocations of the Upper Extremities; Fractures and Dislocations of the Lower Extremities; Sprain, Sprain Fractures, Muscle & Tendon Injuries; Birth Injuries of Motor Skeletal System and Military Surgery.

Forty-three authors who are well recognized in their fields contribute a total of 1063 illustrations to supplement the written words.

Both volumes give a tremendous amount of information. They would be difficult for the inexperienced but those who are interested in Motor-Skeletal Surgery will find much to stimulate and guide them. Material of this sort should raise the standard of surgical procedure.

Faults are present such as a tendency to fatigue the reader by the mass of detail, and the lack of continuity in the basic ideas. There is a wealth of information and these volumes will help solve many problems.

R. F.

Synopsis of Pathology. By W. A. D. ANDERSON. 2nd Ed. Illus. 741 pp. $6.00. *C. V. Mosby Company, St. Louis, Missouri,* 1946.

The purpose of this book as stated in the preface to the first edition is to present pathology in a concise manner for medical and dental students and for the clinician. It is meant to fill a gap between the very elementary manuals of pathology and the "abundant excellent more detailed works." It is the opinion of the reviewer that the stated objectives have been attained with surprising success. It would seem that the only difference between this synopsis and some of the "abundant excellent more detailed works" is a lack of philosophical data rather than of important factual matter. However, a certain amount of fundamental knowledge is sacrificed to conciseness. The author has omitted, for example, in his discussion of dissecting aortic aneurysms, that these lesions may occur in the absence of any intimal tear. He implies that the blood which dissects the aortic wall comes from the lumen of the vessel via a rupture of the intima. The published cases in which there was no intimal tear as well as two such cases personally encountered by the reviewer make it quite clear that in the pathogenesis of dissecting aneurysm, an intimal defect is not always necessary if, indeed it ever is. Furthermore, it is obvious that the blood in the dissection in these cases could not have come from the aortic lumen but must have originated from damaged vasa vasorum.

The Hand-Schüller-Christian syndrome is classified unquestioningly as a disease of lipoid metabolism and no mention is made of the inflammatory aspects of the appearance of its lesions under the microscope. It must, in all fairness, be said that criticisms such as these are equally applicable to almost all the major textbooks of pathology.

The discussion of intestinal obstruction, which is limited to one page is good as

far as it goes. Such subjects as carcinoma of the prostate are very well handled considering the allotted space. A word about hypospadias and other genital abnormalities would be a worthwhile addition.

The organization of the book is such that the first chapters deal with the general aspects of pathology whereas the latter chapters deal with the pathology of special systems. The balance between the space allotted to the various subjects is in the opinion of the reviewer, excellent. Brief summaries of pertinent anatomic and physiological data are given on each subject to help the reader acquire a more complete understanding of the abnormal, but the emphasis in the pathological discussions is a little too much on morphology rather than on pathogenesis and pathological physiology. Tropical diseases and "war medicine" have been given a greater emphasis than in the first edition. The chapters dealing with inflammation, the lung and the nervous system have been extensively revised. Many corrections have been made. For example, the entrance of the poliomyelitis virus by way of the digestive tract, which was omitted from the first edition, has been added in the chapter on virus diseases by Henry Pinkerton.

It is to be hoped that in future editions more and more illustrations will be made from the author's own material. Although a majority of the many black and white and colored photomicrographs and pictures of gross specimens are good or excellent some are in poor focus. As examples of illustrations which could well be improved, the colored photograph of colonies of actinomyces and the black and white photographs of a fibroma and disgerminoma of the ovary may be cited.

One of the greatest attributes of the synopsis other than its well chosen and well presented basic information on pathology, its up to date attitude on many controversial points and the merit of the majority of its illustrations, is its excellent list of modern references.

The medical student or clinician could, of course, not limit his reading in pathology to this synopsis but it makes an excellent text for quick reference and from which to get a start in the general literature of any subject. The author has accomplished his extremely difficult purpose with admirable success and this book is highly recommended for the use for which it is intended.

A. B., III

Textbook of Gynecology. By ARTHUR HALE CURTIS. 5th Ed. Illus. 755 pp. $8.00. *W. B. Saunders Company, Philadelphia, Pennsylvania,* 1946.

The new fifth edition of Dr. Arthus Hale Curtis' Textbook of Gynecology (W. B. Saunders Company) maintains the high standards set by the earlier editions. Many of the chapters have been revised, and of particular note is the chapter on Ovarian Tumors, which is particularly well done. This subject is very thoroughly covered, and even rare tumors as mesonephroma and luteinoma are described. The drawings by Tom Jones, of which those showing the various vaginal plastic operations are especially noteworthy, have been retained from the previous editions. A number of very good photographs and photomicrographs have been added.

Among the new chapters is one giving a very good synopsis of Clinical Endocrinology, and the chapter on Gonorrhoea has been brought up to date by the addition of a discussion of penicillin therapy. Two new topics, Sarcomatous Growth of the Endometrial Stroma, and Hypertrophy of the Uterus have been added to the chapter on Other Tumors of the Uterus. Dr. Curtis has succeeded exceptionally well in keeping his textbook up to date. Although the author's viewpoint is a little old-fashioned at times, his book remains, as it was in the past, one of the very finest. Those of us who have recently returned from the armed forces will find it a very great aid in reviewing the subject of Gynecology.

G. B. B.

BOOKS RECEIVED FOR REVIEW

Bone-Grafting in the Treatment of Fractures. By J. R. ARMSTRONG. 175 pp. $7.00. *Williams & Wilkins Company, Baltimore, Maryland,* 1945.

Carbohydrate Metabolism. By SAMUEL SOSKIN and RACHMIEL LEVINE. 315 pp. $6.00. *The University of Chicago Press, Chicago, Illinois,* 1946.

Diabetes. By HENRY J. JOHN. Illus. 300 pp. $3.25. *The C. V. Mosby Company, St. Louis, Missouri,* 1946.

Diabetic Care in Pictures. By HELEN ROSENTHAL, FRANCES STERN and JOSEPH ROSENTHAL. Illus. 150 pp. $2.00. *J. B. Lippincott Company, Philadelphia, Pennsylvania,* 1946.

Electrocardiography in Practice. By GRAYBIEL and WHITE-ASTON and PAULL. 2nd Ed. Illus. 458 pp. $7.00. *W. B. Saunders Company, Philadelphia, Pennsylvania,* 1946.

English-French and French-English Technical Dictionary. By FRANCIS CUSSET. 591 pp. $5.00. *The Chemical Publishing Company, Brooklyn, New York,* 1946.

Facial Prosthesis. By ARTHUR H. BULBULIAN. Illus. 241 pp. $5.00. *W. B. Saunders Company, Philadelphia, Pennsylvania,* 1945.

Fees and Fee Bills: Some Economic Aspects of Medical Practice in Nineteenth Century America. By GEORGE ROSEN. 93 pp. $1.50. *The Johns Hopkins Press, Baltimore, Maryland,* 1946.

Human Torluosis. By LEONARD B. COX and JEAN C. TOLHURST. 148 pp. $2.51. *Oxford University Press, New York, New York,* 1946.

The Modern Treatment of Diabetes Mellitus. By WILLIAM S. COLLENS and LOUIS C. BOAS. 514 pp. $8.50. *Charles C Thomas, Springfield, Illinois,* 1946.

Narco-Analysis. By J. STEPHEN HORSLEY. 134 pp. $2.50. *Oxford University Press, New York, New York,* 1943.

Neurosis and the Mental Health Services. 218 pp. $5.00. *Oxford University Press, New York, New York,* 1946.

New and Nonofficial Remedies. 770 pp. $1.50. *American Medical Association, Chicago, Illinois,* 1946.

New Aspects of John and William Hunter. By JANE M. OPPENHEIMER. 188 pp. $6.00. *Henry Schuman, New York, New York,* 1946.

Peripheral Vascular Diseases. By EDGAR V. ALLEN, NELSON W. BARKER and EDGAR A. HINES, JR. Illus. 871 pp. $10.00. *W. B. Saunders, Philadelphia, Pennsylvania,* 1946.

Sex Problems of the Returned Veteran. By HOWARD KITCHING. 124 pp. $1.50. *Emerson Books, Incorporated, New York, New York,* 1946.

The Surgery of Repair: Injuries and Burns. D. N. MATTHEWS. Illus. 386 pp. $10.00. *Charles C Thomas, Springfield, Illinois,* 1943.

Traumatic Injuries of Facial Bones. By JOHN B. ERICH and LOUIE T. AUSTIN. Illus. 600 pp. $6.00. *W. B. Saunders Company, Philadelphia, Pennsylvania,* 1944.

William Beaumont's Formative Years. Illus. 87 pp. $6.00. *Henry Schuman, New York, New York,* 1946.

Women in Industry: Their Health and Efficiency. By ANNA M. BAETZER. 344 pp. $4.00. *W. B. Saunders Company, Philadelphia, Pennsylvania,* 1946.